Endocrine system

Meaning and importance
Hormones
Pituitary gland
Pineal gland
Thyroid gland
Parathyroid gland
Thymus gland
Pancreas
Adrenal glands
Gonadal hormone
Hormones from kidney, heart, placenta and GI tract
General mechanism of hormone action
Hormones and diseases

Endocrine system

- Regulatory system of ductless glands
- Secretes chemical messengers called **Hormones**
- Circulates within the body via bloodstream to affect distant organs
- Ductless glands = **Endocrine glands**
- Secrete hormones directly into the bloodstream for distribution throughout the body
- Do not only include all endocrine glands but also hormone secreting cells

Ex

- H

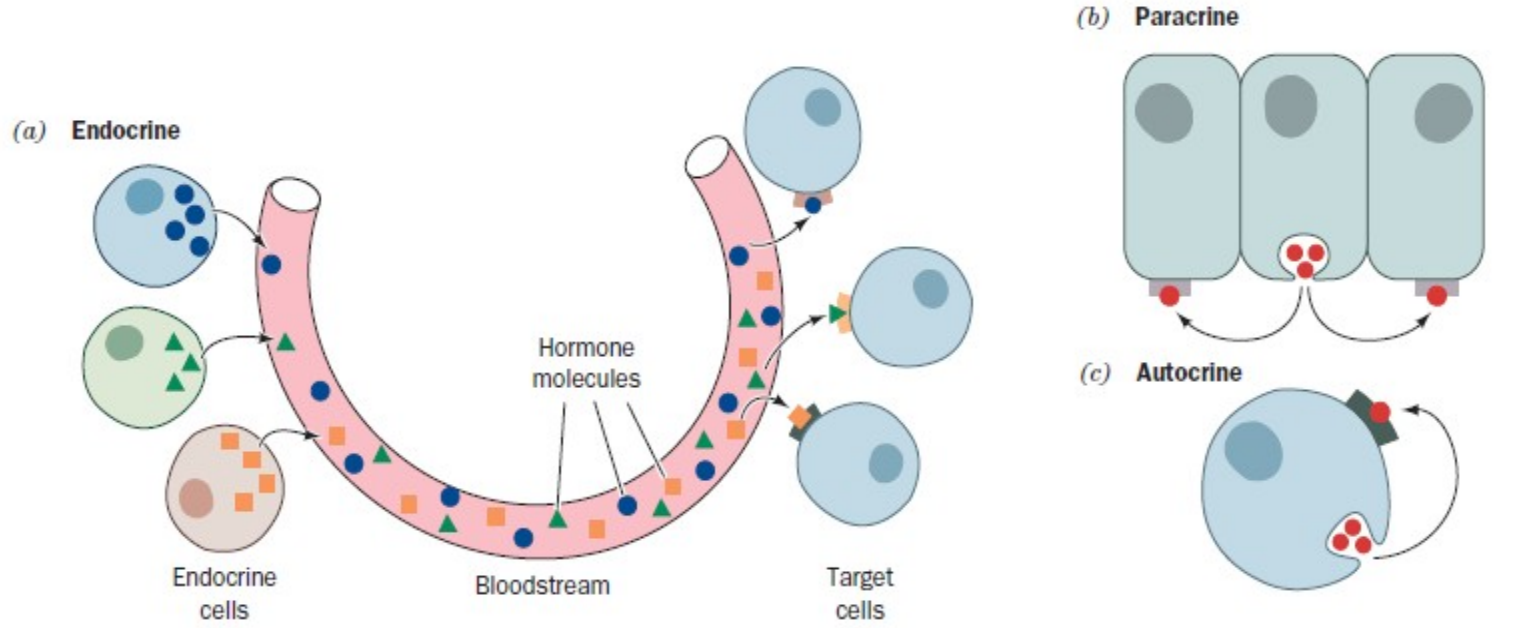

Figure 19-1 **Classification of hormones.** Hormonal communications are classified according to the distance over which the signal acts: (*a*) endocrine signals are directed at distant cells through the intermediacy of the bloodstream, (*b*) paracrine signals are directed at nearby cells, and (*c*) autocrine signals are directed at the cell that produced them.

Source: Donald Voet, Judith G Voet (2011), John Wiley & Sons. Inc, 4th

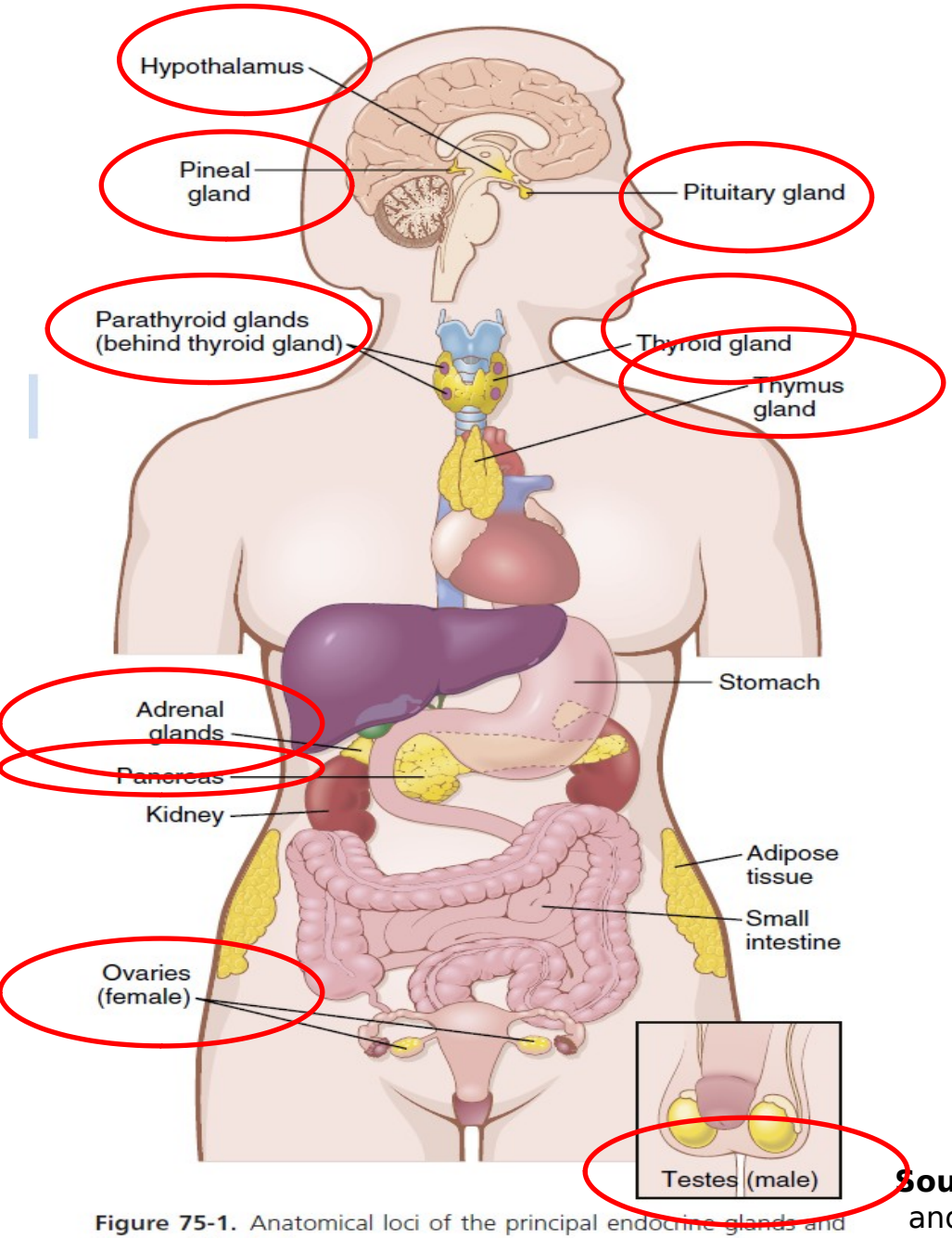

Figure 75-1. Anatomical loci of the principal endocrine glands and tissues of the body.

Major Endocrine glands of Body:

1. Hypothalamus
2. Pituitary gland
3. Pineal gland
4. Thyroid gland
5. Parathyroid gland
6. Thymus gland
7. Pancreas
8. Adrenal glands
9. Ovaries
10. Testes

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 867

Rashmi MG

Source: David L Nelson, Michael M Cox, Lehninger Principle of Biochemistry, W H Freeman Publishers, 4th edition Page no.885

Hormones

- Chemical messengers
- Signal molecules
- Endocrine signal molecules = released by cells of endocrine glands into the blood and transported to a distantly located target organs
- Low molecular weight
- Chemically heterogeneous non nutrient substances which is produced in trace amounts and acts as a intercellular signal molecules
- Only specific target cells can respond to each hormones
- Because they have receptors for binding with particular hormone

Hormonal signalling

1. Biosynthesis of hormones in particular tissue
2. Storage and secretion of the hormone
3. Transport of the hormone to target cell
4. Recognition of the hormone by an associated cell membrane or intracellular receptor protein

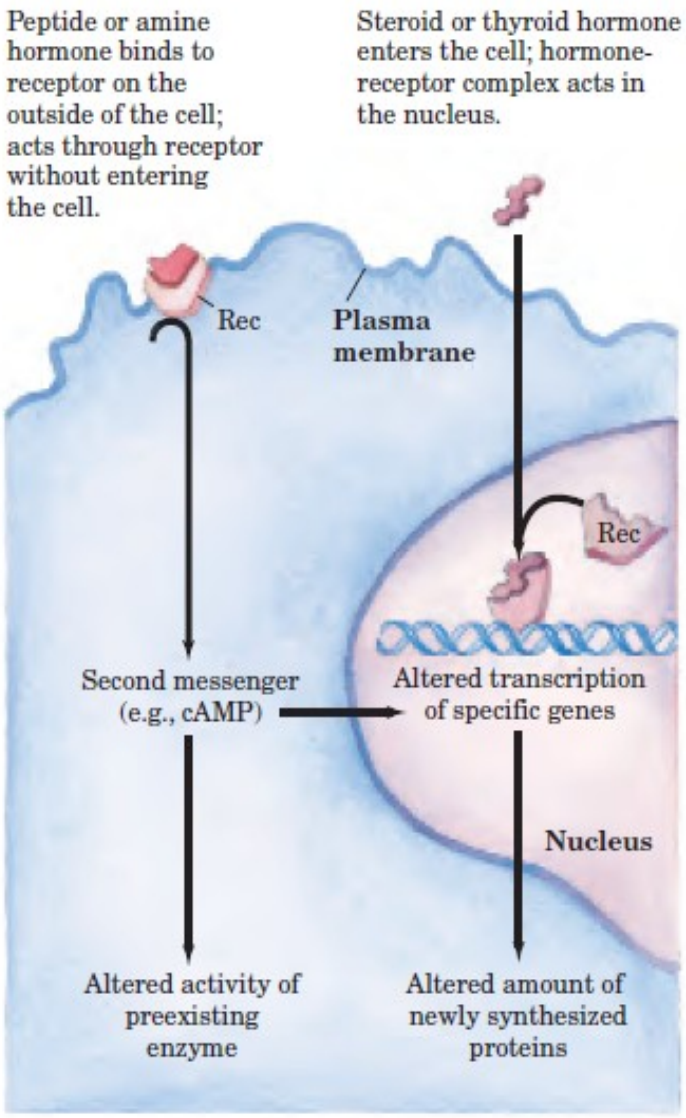
Peptide or amine hormone binds to receptor on the outside of the cell; acts through receptor without entering the cell.
Steroid or thyroid hormone enters the cell; hormone-receptor complex acts in the nucleus.
Rec
Plasma membrane
Rec
Second messenger (e.g., cAMP)
Altered transcription of specific genes
Nucleus
Altered activity of preexisting enzyme
Altered amount of newly synthesized proteins

FIGURE 23–4 Two general mechanisms of hormone action. The peptide and amine hormones are faster acting than steroid and thyroid hormones.

Rashmi MG

Classification of hormones

Lipid-Soluble Hormones

Steroid Hormones- derived from **cholesterol**

Thyroid hormones- (**T_3** and **T_4-** synthesized by attaching iodine to **Tyrosine**

Benzene ring of tyrosine makes **T_3** and **T_4** lipid soluble

Glycoprotein hormones

Hormones with attached **carbohydrate groups**

Ex. **TSH**

Water-Soluble Hormones

Amine hormones- synthesized by decarboxylation / modification of certain amino acids

Ex. **Catecholamines**, **Epinephrine**, **Norepinephrine** and **Dopamine-** synthesized by modifying amino acid Tyrosine

Histamine- synthesized from **Histidine** by Mast cells and platelets

Serotonin and **Melatonin-** derived from Tryptophan

Peptide hormones- consists of chains of 3-49 amino acids

Ex. **ADH** and **Oxytocin**

Protein hormones- consists of 50-200 amino acids

Ex. **Growth hormones** and **Insulin**

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Table 19-1 Some Human Hormones

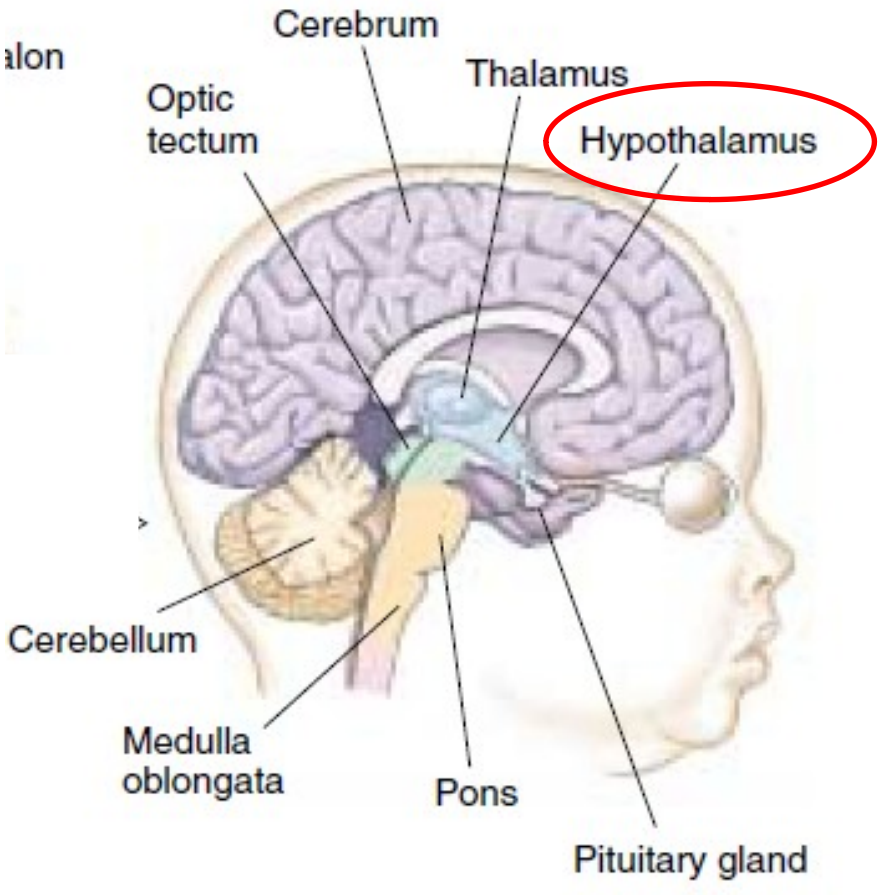
Hormone	Origin	Major Effects
Polypeptides		
Corticotropin-releasing factor (CRF)	Hypothalamus	Stimulates ACTH release
Gonadotropin-releasing factor (GnRF)	Hypothalamus	Stimulates FSH and LH release
Thyrotropin-releasing factor (TRF)	Hypothalamus	Stimulates TSH release
Growth hormone-releasing factor (GRF)	Hypothalamus	Stimulates growth hormone release
Somatostatin	Hypothalamus	Inhibits growth hormone release
Adrenocorticotropic hormone (ACTH)	Adenohypophysis	Stimulates the release of adrenocorticosteroids
Follicle-stimulating hormone (FSH)	Adenohypophysis	In ovaries, stimulates follicular development, ovulation, and estrogen synthesis; in testes, stimulates spermatogenesis
Luteinizing hormone (LH)	Adenohypophysis	In ovaries, stimulates oocyte maturation and follicular synthesis of estrogens and progesterone; in testes, stimulates androgen synthesis
Chorionic gonadotropin (CG)	Placenta	Stimulates progesterone release from the corpus luteum
Thyrotropin (TSH)	Adenohypophysis	Stimulates T_3 and T_4 release
Somatotropin (growth hormone)	Adenohypophysis	Stimulates growth and synthesis of somatomedins
Met-enkephalin	Adenohypophysis	Opioid effects on central nervous system
Leu-enkephalin	Adenohypophysis	Opioid effects on central nervous system
β-Endorphin	Adenohypophysis	Opioid effects on central nervous system
Vasopressin	Neurohypophysis	Stimulates water resorption by kidney and increases blood pressure
Oxytocin	Neurohypophysis	Stimulates uterine contractions
Glucagon	Pancreas	Stimulates glucose release through glycogenolysis and stimulates lipolysis
Insulin	Pancreas	Stimulates glucose uptake through gluconeogenesis, protein synthesis, and lipogenesis
Gastrin	Stomach	Stimulates gastric acid and pepsinogen secretion
Secretin	Intestine	Stimulates pancreatic secretion of HCO_3^-
Cholecystokinin (CCK)	Intestine	Stimulates gallbladder emptying and pancreatic secretion of digestive enzymes and HCO_3^-
Gastric inhibitory peptide (GIP)	Intestine	Inhibits gastric acid secretion and gastric emptying; stimulates pancreatic insulin release
Parathyroid hormone	Parathyroid	Stimulates Ca^{2+} uptake from bone, kidney, and intestine
Calcitonin	Thyroid	Inhibits Ca^{2+} uptake from bone and kidney
Somatomedins	Liver	Stimulates cartilage growth; have insulinlike activity

Steroids		
Glucocorticoids	Adrenal cortex	Affect metabolism in diverse ways, decrease inflammation, increase resistance to stress
Mineralocorticoids	Adrenal cortex	Maintain salt and water balance
Estrogens	Gonads	Maturation and function of secondary sex organs, particularly in females
Androgens	Gonads	Maturation and function of secondary sex organs, particularly in males; male sexual differentiation
Progestins	Ovaries and placenta	Mediate menstrual cycle and maintain pregnancy
Vitamin D	Diet and sun	Stimulates Ca^{2+} absorption from intestine, kidney, and bone
Amino Acid Derivatives		
Epinephrine	Adrenal medulla	Stimulates contraction of some smooth muscles and relaxes others, increases heart rate and blood pressure, stimulates glycogenolysis in liver and muscle, stimulates lipolysis in adipose tissue
Norepinephrine	Adrenal medulla	Stimulates arteriole contraction, decreases peripheral circulation, stimulates lipolysis in adipose tissue
Triiodothyronine (T_3)	Thyroid	General metabolic stimulation
Thyroxine (T_4)	Thyroid	General metabolic stimulation

Source: Donald Voet, Judith G Voet (2011), John Wiley & Sons. Inc, 4th Edition, Page no. 973

Hypothalamus

- **Location**: Base of **Diencephalon** of the **forebrain**
- Considered as a **Neuroendocrine** structure
- Secretions is called **Neurohormone**
- These hormones originate in Hypothalamic neurons, pass through axons and are released from the nerve endings reaches the pituitary gland through a portal circulatory system and regulate the function of the Anterior pituitary
- Also posterior pituitary is under direct neural regulation of Hypothalamus
- **Main function:** Regulate the synthesis and secretion of pituitary hormones
- There are 2 types of hormones (released by Hypothalamus based on action) –
  - **Releasing Hormones**



Source: Raven Johnson, Biology, 6th Edition, Page no.1090

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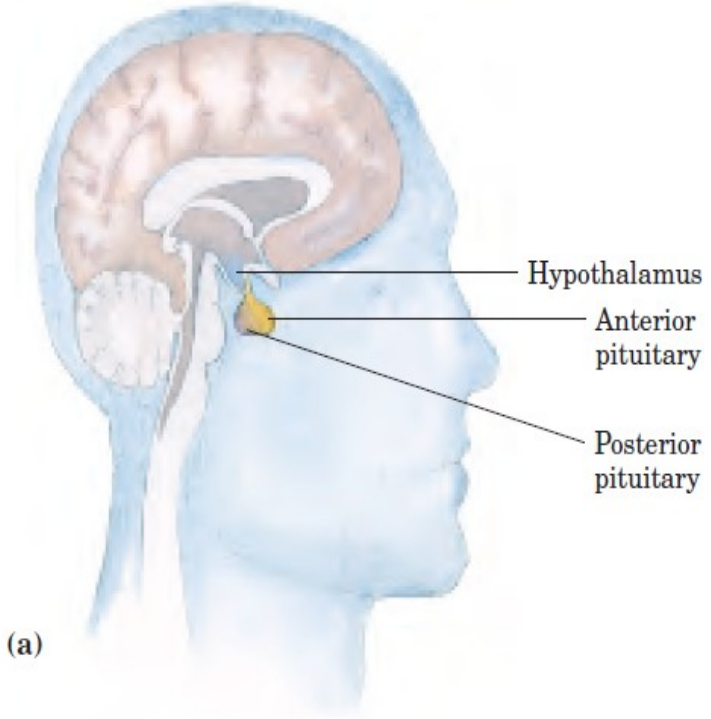


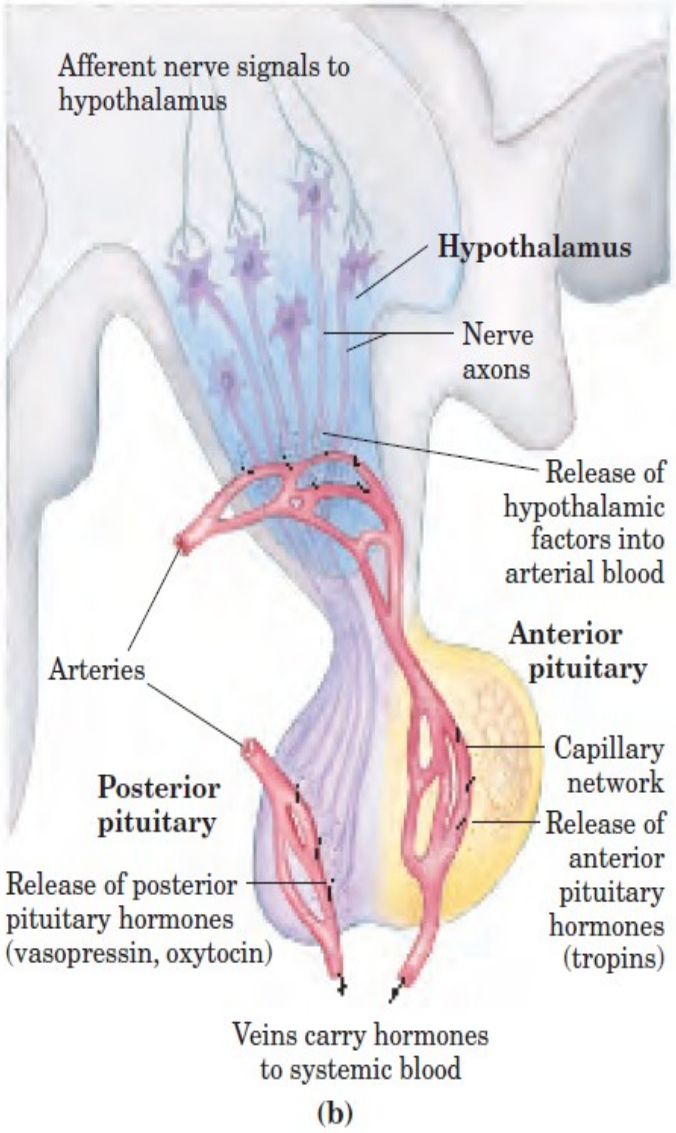
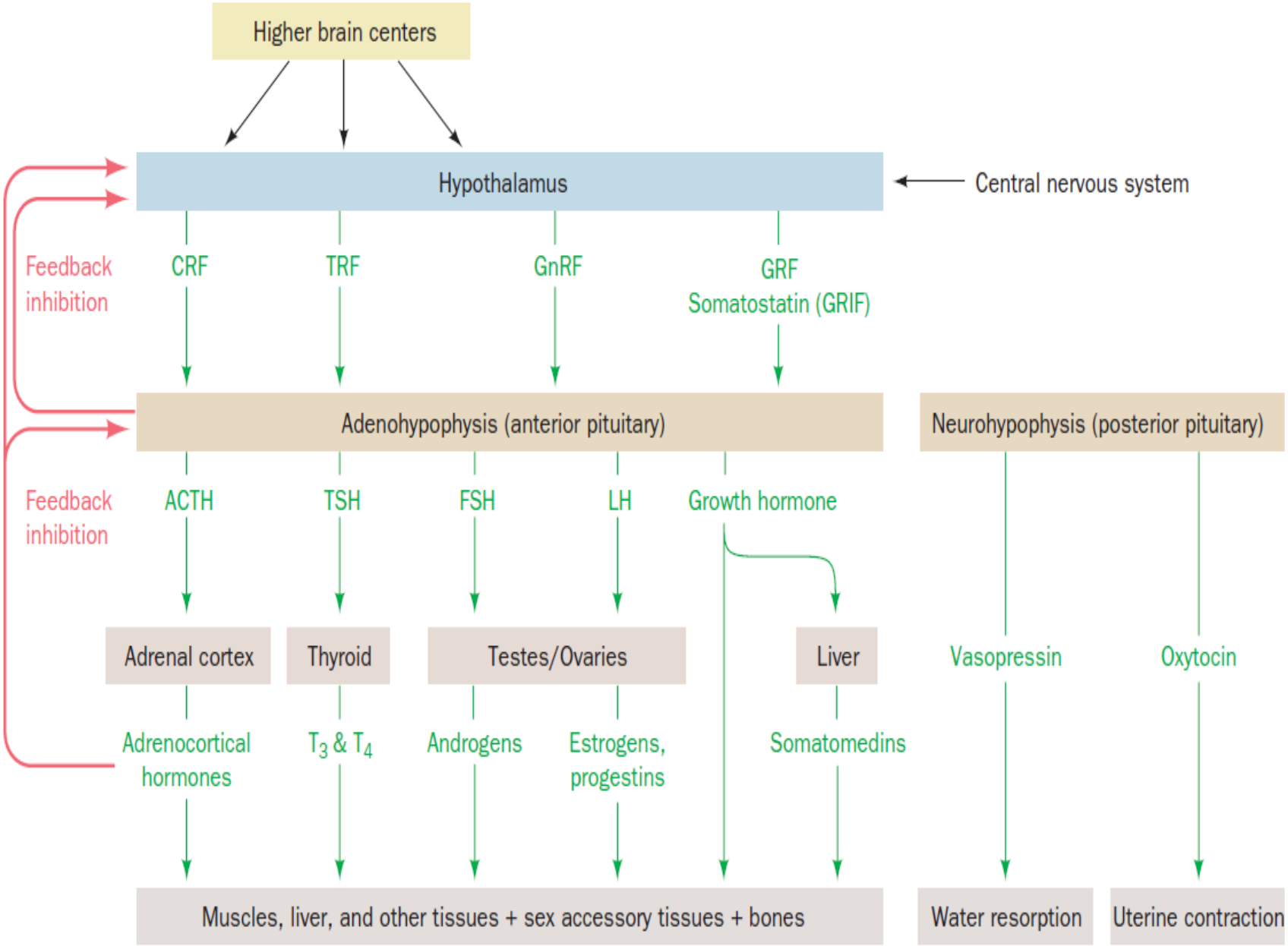


FIGURE 23–9 Neuroendocrine origins of hormone signals. (a) Location of the hypothalamus and pituitary gland. (b) Details of the hypothalamus-pituitary system. Signals from connecting neurons stimulate the hypothalamus to secrete releasing factors into a blood vessel that carries the hormones directly to a capillary network in the anterior pituitary. In response to each hypothalamic releasing factor, the anterior pituitary releases the appropriate hormone into the general circulation. Posterior pituitary hormones are synthesized in neurons arising in the hypothalamus, transported along axons to nerve endings in the posterior pituitary, and stored there until released into the blood in response to a neuronal signal.



Source: Donald Voet, Judith G Voet (2011), John Wiley & Sons. Inc, 4th

Releasing hormones

Thyrotropin-releasing hormone
- **TRH**
- Also called **Thyrotropin-releasing factor (TRF)**
- Peptide hormone
- Produced by **Hypothalamus** and travels to pituitary via **Hypophyseal portal system**
- Stimulates the release of **Thyroid-stimulating hormone** (TSH) and **Prolactin** by **Anterior pituitary**

Gonadotropin-releasing hormone
- **GnRH**
- Peptide hormone
- Responsible for the release of **follicle stimulating hormones (FSH)** and **Luteinizing hormone (LH)** from the **anterior pituitary**

Corticotropin –releasing hormone
- **CRH**
- Originally names **Corticotropin- releasing factor (CRF**) also called **corticoliberin**
- Polypeptide hormone and neurotransmitter
- Stimulates the secretion of **ACTH** from **pituitary**

Growth hormone- Releasing hormones
- **GHRH**
- Known as **growth hormone- releasing factor (GRF/ GHRF)**
- Peptide hormones produced by **Hypothalamus**
- Released from **neurosecretory nerve terminals** and carried by the **hypothalamo-hypophyseal portal circulation** to the **anterior pituitary gland** where it stimulates the synthesis and release of **growth hormones (GH)**

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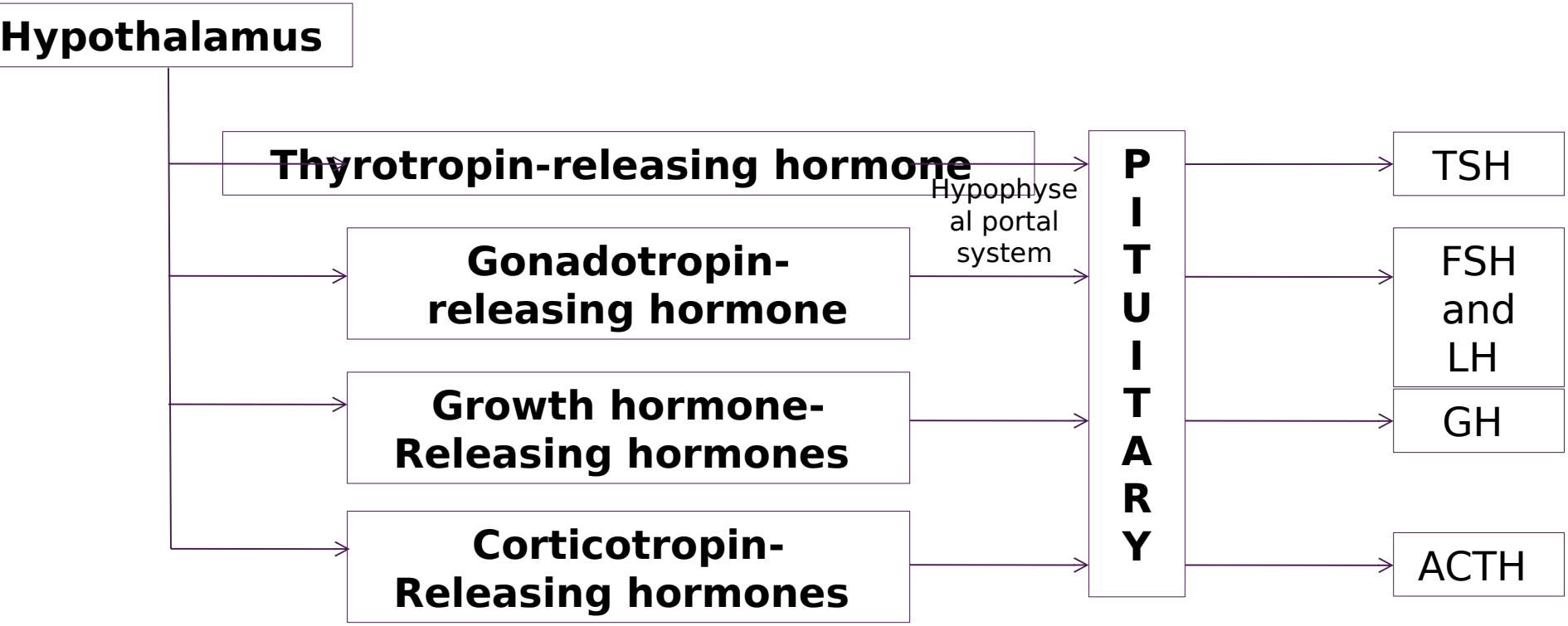

Hypothalamus
Thyrotropin-releasing hormone
Gonadotropin-releasing hormone
Growth hormone-Releasing hormones
Corticotropin-Releasing hormones
Hypophyseal portal system
PITUITARY
TSH
FSH and LH
GH
ACTH

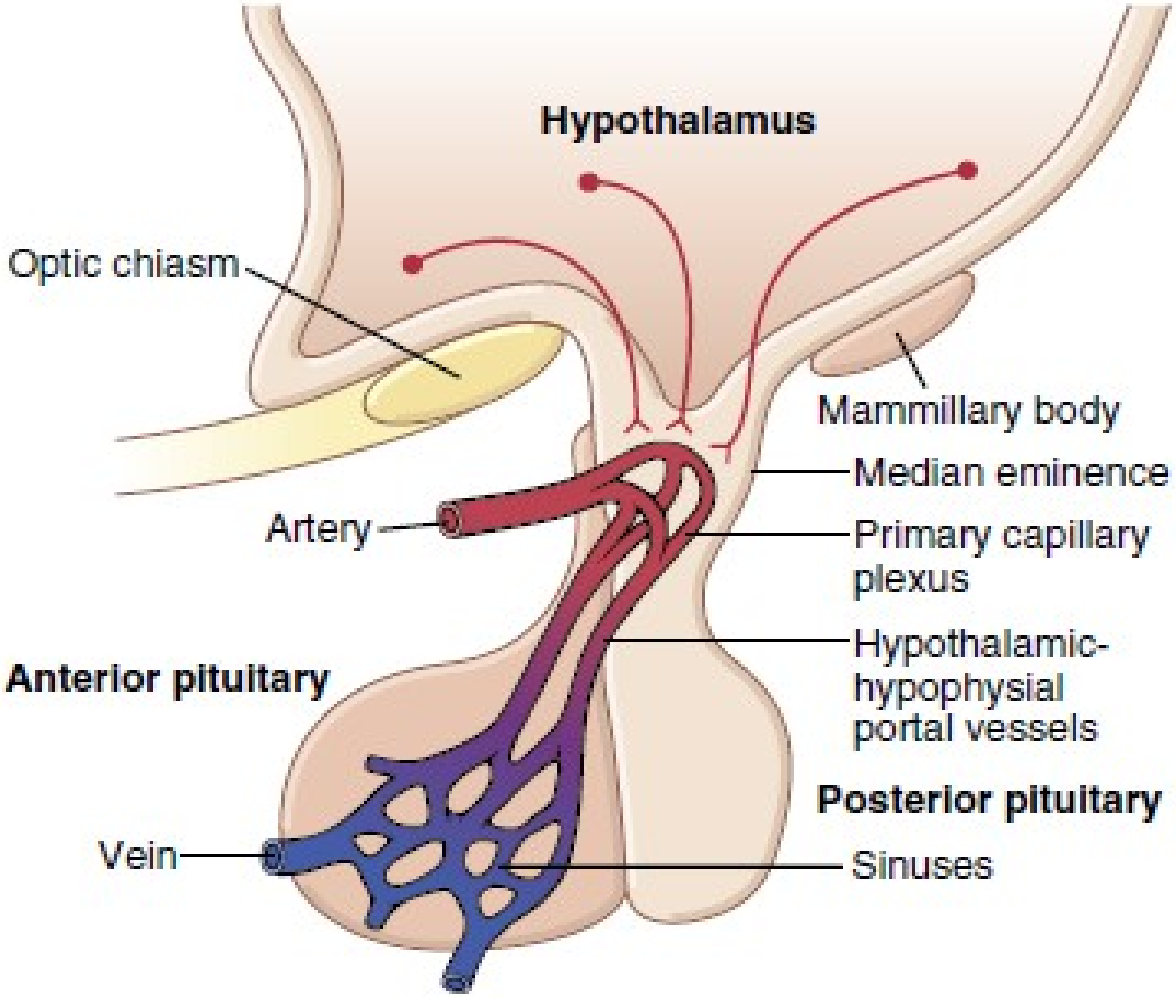


Figure 76-4. Hypothalamic-hypophysial portal system.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 881

Inhibiting hormones

Prolactin- inhibiting hormone

- **PIH**
- **Dopamine**
- Inhibits the release of **Prolactin** from **Anterior Pituitary**
- In brain, dopamine function as a neurotransmitter, activating dopamine receptors

Somatostatin

- **Hypothalamic** hormone
- Also called as **Growth Hormone- Inhibiting Hormone (GHIH)**
- Peptide hormone
- 2 active forms of Somatostatin are synthesized – **Somatostatin-14** and **Somatostatin-28** (number reflecting the number of amino acid residues)
- Somatostatin-14= abundant in hypothalamus and endocrine pancreas
- Somatostatin-28= most abundant in the intestine

Functions:

- Inhibits the release of **Growth Hormone , Thyroid-stimulating Hormone**
- Suppress the release of **Gastrointestinal Hormones, Pancreatic Hormones Insulin** and **Glucagon**, exocrine secretory action of **Pancreas**
- Oppose the effects of **GHRH**

Pituitary gland

- Also called as Hypophysis
- **Location**: **Sella Turcica** of sphenoid bone, attached to the **Hypothalamus** by **Infundibulum**
- Consists of 2 major parts:
  - Large anterior pituitary (**Adenohypophysis**)
  - Small posterior

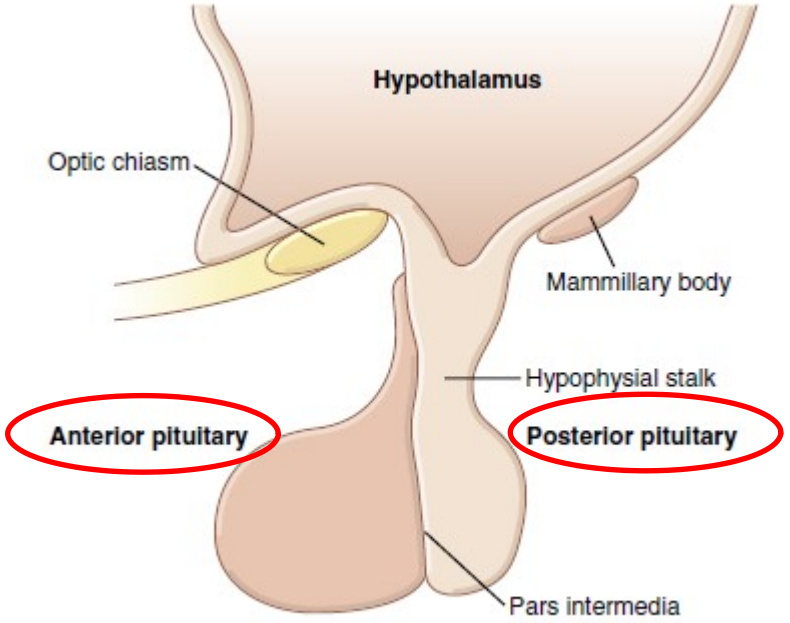
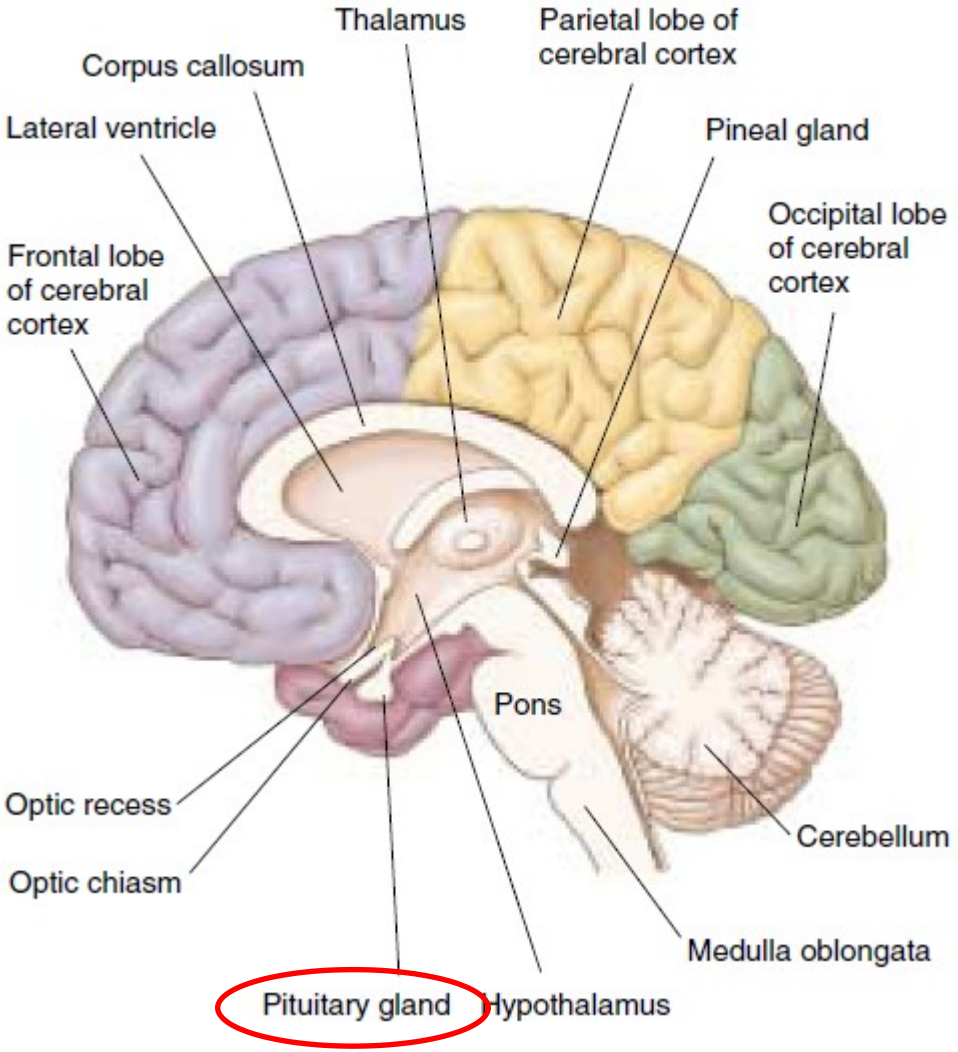


Figure 76-1. Pituitary gland.



Source: Raven Johnson, Biology, 6th Edition, Page no.1093

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier

Table 76-1 Cells and Hormones of the Anterior Pituitary Gland and Their Physiological Functions

Cell	Hormone	Chemistry	Physiological Action
Somatotropes	Growth hormone (GH) (somatotropin)	Single chain of 191 amino acids	Stimulates body growth; stimulates secretion of insulin-like growth factor-1; stimulates lipolysis; inhibits actions of insulin on carbohydrate and lipid metabolism
Corticotropes	Adrenocorticotropic hormone (ACTH) (corticotropin)	Single chain of 39 amino acids	Stimulates production of glucocorticoids and androgens by the adrenal cortex; maintains size of zona fasciculata and zona reticularis of cortex
Thyrotropes	Thyroid-stimulating hormone (TSH) (thyrotropin)	Glycoprotein of two subunits, α (89 amino acids) and β (112 amino acids)	Stimulates production of thyroid hormones by thyroid follicular cells; maintains size of follicular cells
Gonadotropes	Follicle-stimulating hormone (FSH)	Glycoprotein of two subunits, α (89 amino acids) and β (112 amino acids)	Stimulates development of ovarian follicles; regulates spermatogenesis in the testis
	Luteinizing (LH) hormone	Glycoprotein of two subunits, α (89 amino acids) and β (115 amino acids)	Causes ovulation and formation of the corpus luteum in the ovary; stimulates production of estrogen and progesterone by the ovary; stimulates testosterone production by the testis
Lactotropes-mammotropes	Prolactin (PRL)	Single chain of 198 amino acids	Stimulates milk secretion and production

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 881

Anterior pituitary

- Called as **Adenohypophysis**
- Secretes 7 hormones and release of these hormones are stimulated by Releasing hormones and inhibited by Inhibiting hormones
- Consists of 2 portions: (often merged)
- **Pars distalis** – secretes **GH**, **PRL**, **TSH**, **ACTH**, **LH**, **FSH**
- **Pars intermedia** – secretes **MSH**

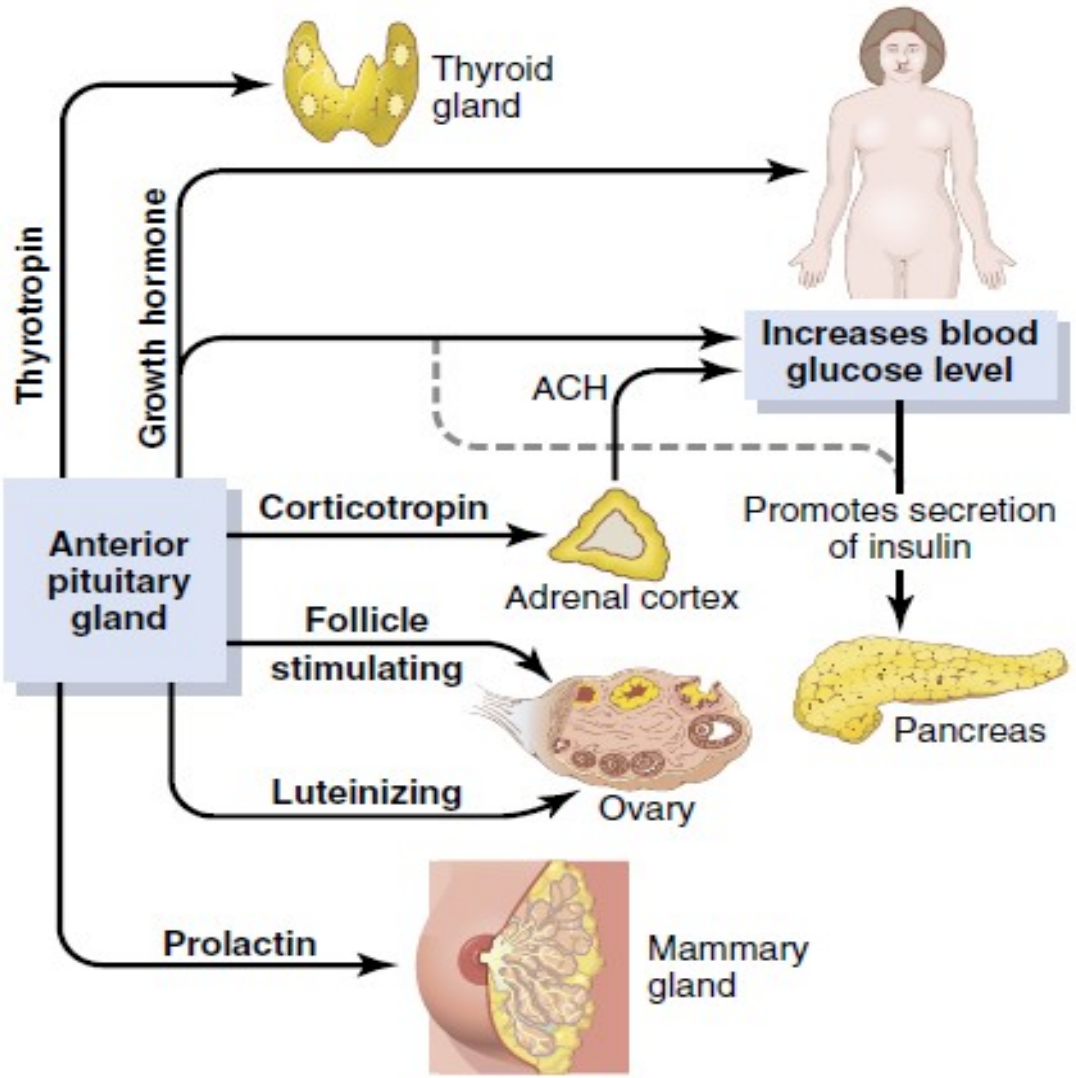


Figure 76-2. Metabolic functions of the anterior pituitary hormones. ACH, Adrenal corticosteroid hormones.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 880

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Growth hormones (GH)

- Also called as **Somatotropin**
- Promotes the movement of amino acids into cells and incorporation of these amino acids into proteins = **promotes overall tissue and organ growth**
- Exerts cell division stimulating effect (mitogenic effect)= indirectly through mediation of mitogen (called **Insulin-like Growth Factor-i**)
- Influence of **GH→ IGF-I** secreted by liver → enters the blood → functions as Hormone

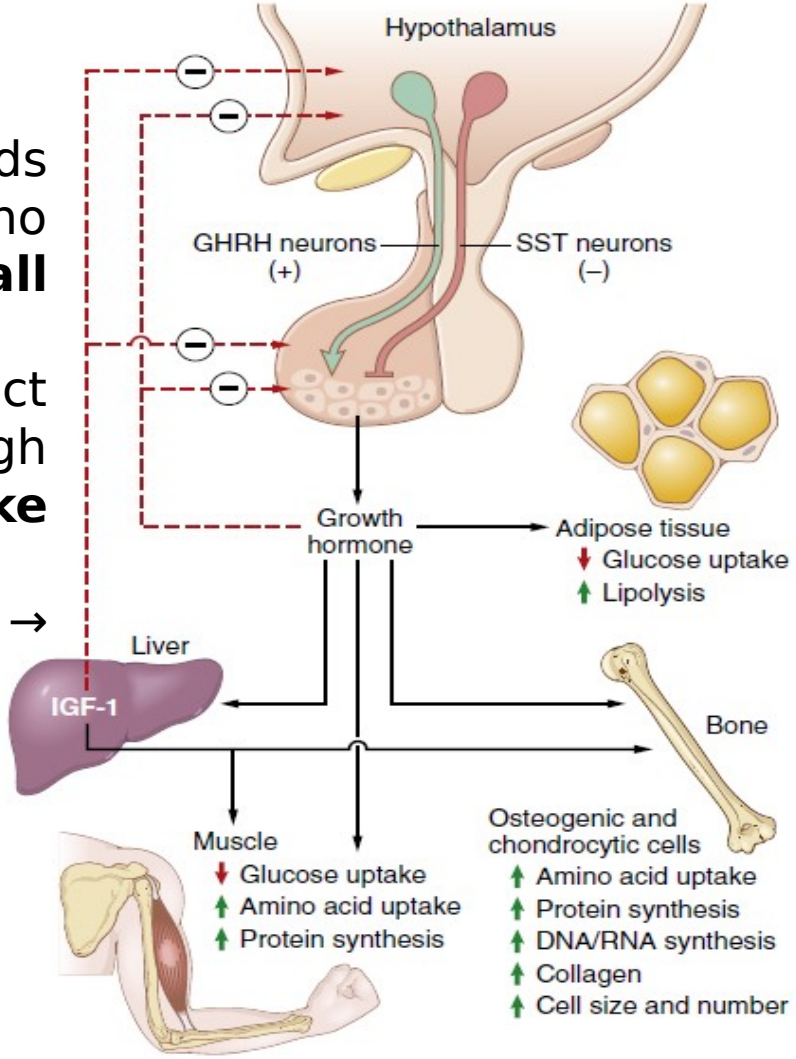

Figure 76-6. Effects of growth hormone and insulin-like growth factor -1 (IGF-1) on growth and metabolism. Growth hormone secretion is stimulated by growth hormone–releasing hormone (GHRH) and inhibited by somatostatin (SST), as well as negative feedback effects of growth hormone and IGF-1 on the anterior pituitary gland and the hypothalamic neurons. Other factors that control growth hormone secretion are described in Table 76-3.

Table 76-3 Factors That Stimulate or Inhibit Secretion of Growth Hormone

Stimulate Growth Hormone Secretion	Inhibit Growth Hormone Secretion
Decreased blood glucose level	Increased blood glucose level
Decreased blood free fatty acid levels	Increased blood free fatty acid levels
Increased blood amino acid levels (arginine)	Aging
Starvation or fasting, protein deficiency	Obesity
Trauma, stress, excitement	Growth hormone inhibitory hormone (somatostatin)
Exercise	Growth hormone (exogenous)
Testosterone, estrogen	Insulin-like growth factors (somatomedins)
Deep sleep (stages 2 and 4)	
Growth hormone–releasing hormone	
Ghrelin	

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 883

Prolactin

- **PRL**
- Peptide hormones
- Synthesized and secreted by **Adenohypophysis**
- Stimulates mammary glands to produce milk (**lactation**)
- Increased serum concentration of Prolactin during pregnancy→ enlargement of mammary glands in breast → Increase production of milk
- **High levels** of **progesterone** (during pregnancy) = act directly on breasts to stop its lactogenic effect
- **Low progesterone level**(fall by end of pregnancy) = Milk production starts
- Also gives negative feedback to hypothalamus = to stop secretion **GnRH** → Decreases secretion of FSH and **LH** from anterior pituitary → Stops ovulation during pregnancy

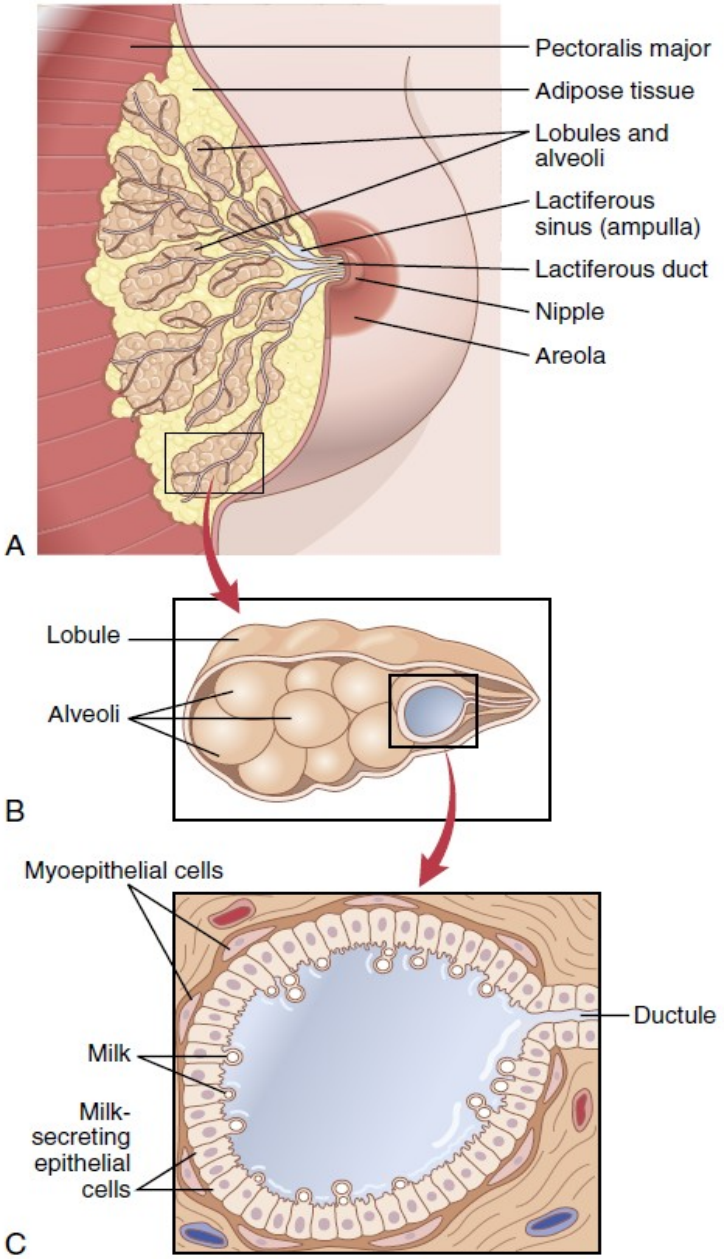

Figure 83-11. A, The breast and its secretory lobules, alveoli, and lactiferous ducts (milk ducts) that constitute its mammary gland. **B,** The enlargements show a lobule and milk-secreting cells (**C**) of an alveolus.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 1003

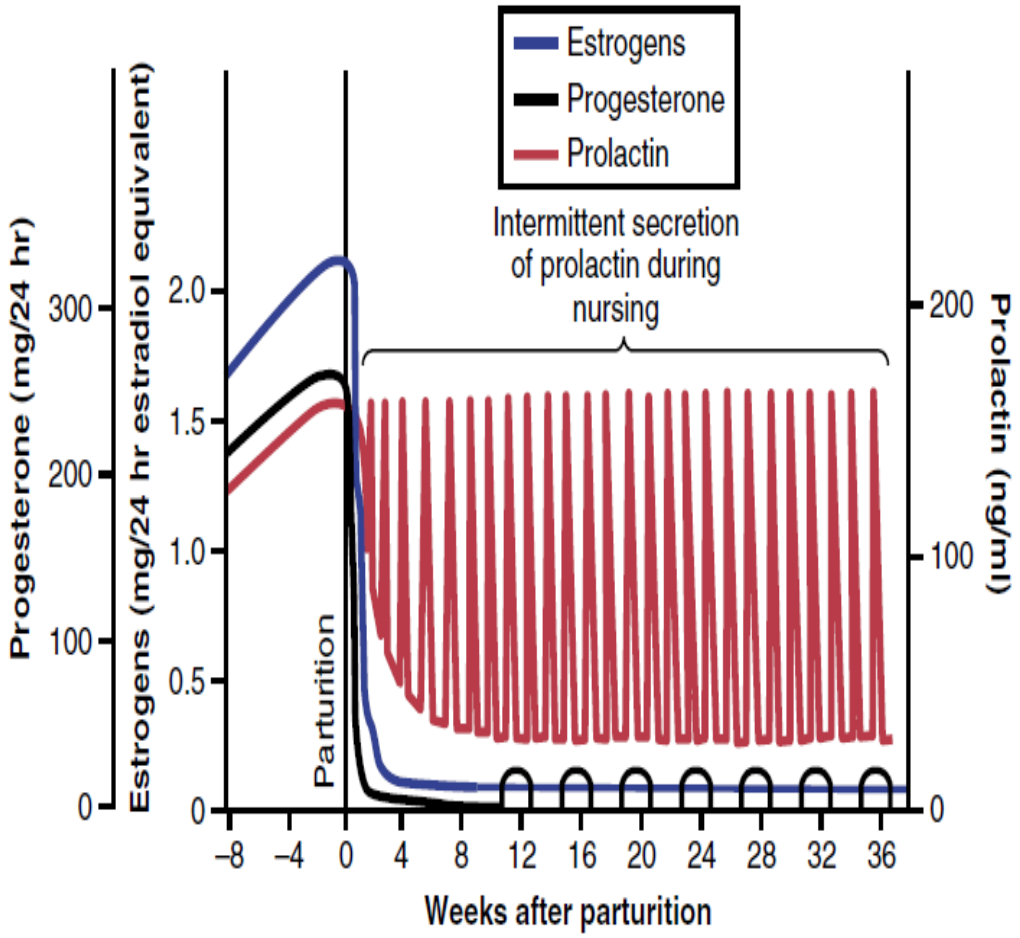

Figure 83-12. Changes in rates of secretion of estrogens, progesterone, and prolactin for 8 weeks before parturition and 36 weeks thereafter. Note especially the decrease of prolactin secretion back to basal levels within a few weeks after parturition, but also the intermittent periods of marked prolactin secretion (for about 1 hour at a time) during and after periods of nursing.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 1004

Adrenocorticotropic hormone

- **ACTH**
- Also called as **Corticotropin**
- Polypeptide hormones
- Synthesized in the **anterior lobe of the pituitary gland** in response to **CRH** released by **Hypothalamus**
- Stimulates synthesis and secretion of **steroid hormones** called **Corticosteroids** from the **adrenocortical cells** of **adrenal cortex**

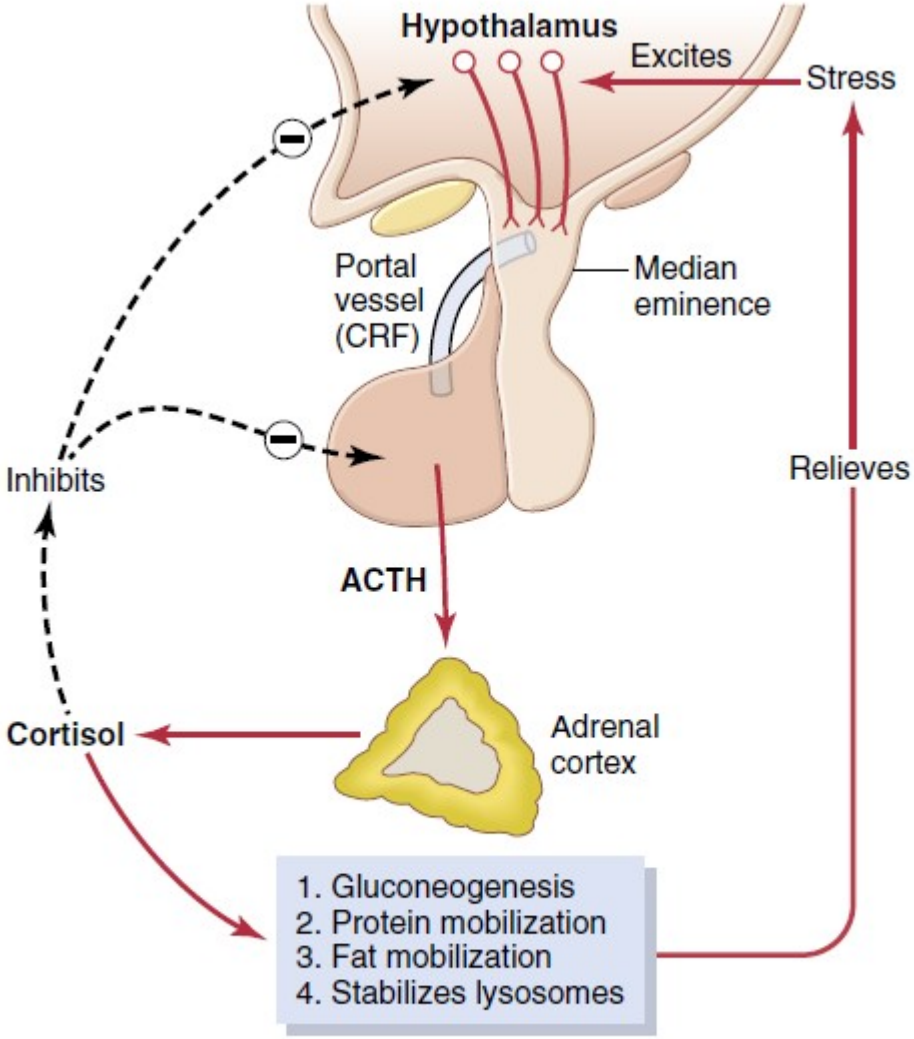

Figure 78-8. Mechanism for regulation of glucocorticoid secretion. ACTH, Adrenocorticotropic hormone; CRF, corticotropin-releasing factor.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 916

Thyroid stimulating hormone

- **TSH**
- **Thyrotropin**
- Stimulates the synthesis and secretion of **thyroid hormones** from the thyroid gland

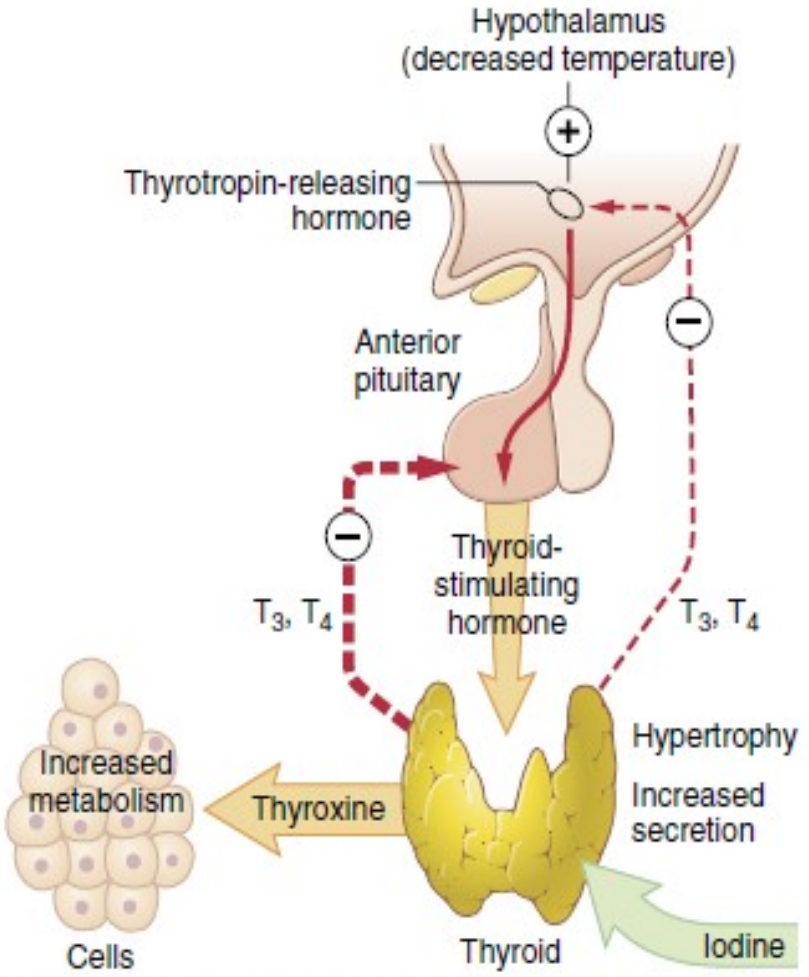

Figure 77-7 Regulation of thyroid secretion. T_3, triiodothyronine; T_4, thyroxine.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 899

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Follicle stimulating hormone

- **FSH**
- Stimulates gamete (ova and sperm) production in both sexes
- **In females,**
  = stimulates growth and development of ovarian follicles → eggs or ova develops
  =Promotes secretion of estrogen by ovaries
- **In males**
  = Targets seminiferous tubules in testes → stimulates sperm production

Luteinizing hormone

- **In females**
  Triggers ovulation from **Graafian Follicle** = release of secondary oocyte by an ovary
  Stimulates formation of **Corpus Luteum**
- Secretion of **Progesterone**
- **In males**
- Called as **Interstitial Cell Stimulating Hormone (ICSH)**
  Stimulates cells in testes – secretes **Testosterone**

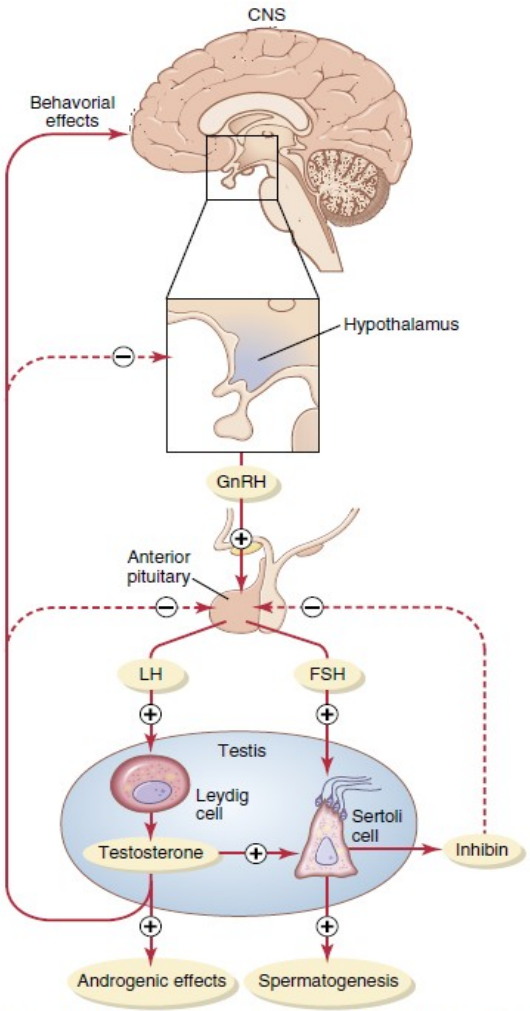


Figure 81-10 Feedback regulation of the hypothalamic-pituitary-testicular axis in males. Stimulatory effects are shown by *plus signs*, and negative feedback inhibitory effects are shown by *minus signs*. CNS, Central nervous system; FSH, follicle-stimulating hormone; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone.

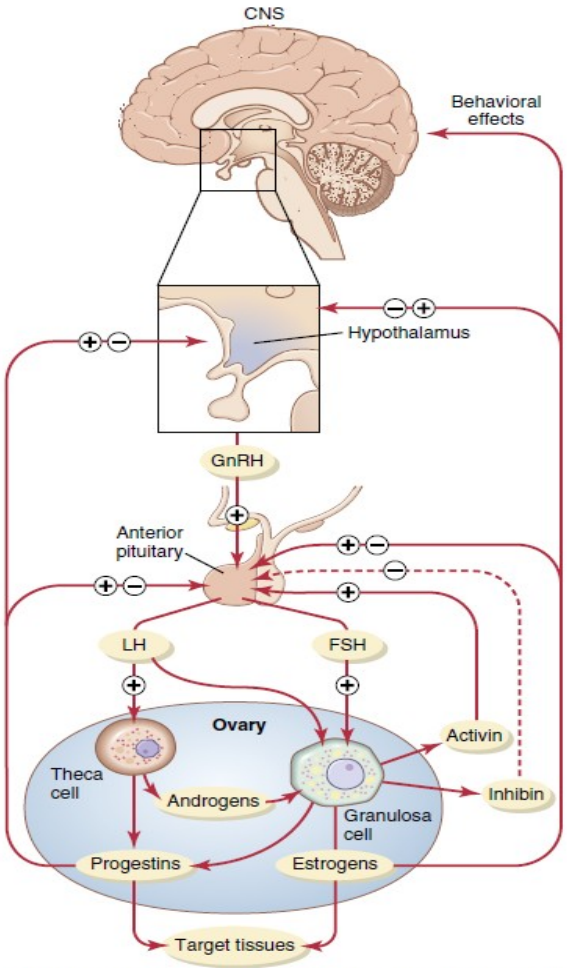


Figure 82-11. Feedback regulation of the hypothalamic-pituitary-ovarian axis in females. Stimulatory effects are shown by plus signs (+); negative feedback inhibitory effects are shown by minus signs (–). Estrogens and progestins exert negative and positive feedback effects on the anterior pituitary and hypothalamus, depending on the stage of the ovarian cycle. Inhibin has a negative feedback effect on the anterior pituitary, whereas activin has the opposite effect, stimulating follicle-stimulating hormone (FSH) secretion by the anterior pituitary. CNS, Central nervous system; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 969, 985

Posterior pituitary

- **Neurohypophysis**
- Does not synthesize much hormones
- Stores and releases two hormones – **Oxytocin** and **Vasopressin**
- These are actually synthesized by **Hypothalamus** and are transported axonally to **Neurohypophysis**

Oxytocin

- **Pitocin**
- Stimulates uterine contractions at the time of child birth
- Stimulates the release of milk from the mammary glands by causing surrounding cells to contract
- After birth, stimulation of breast by the infant feeding stimulates the posterior pituitary to produce Oxytocin
- Often called **Birth hormone**

Vasopressin

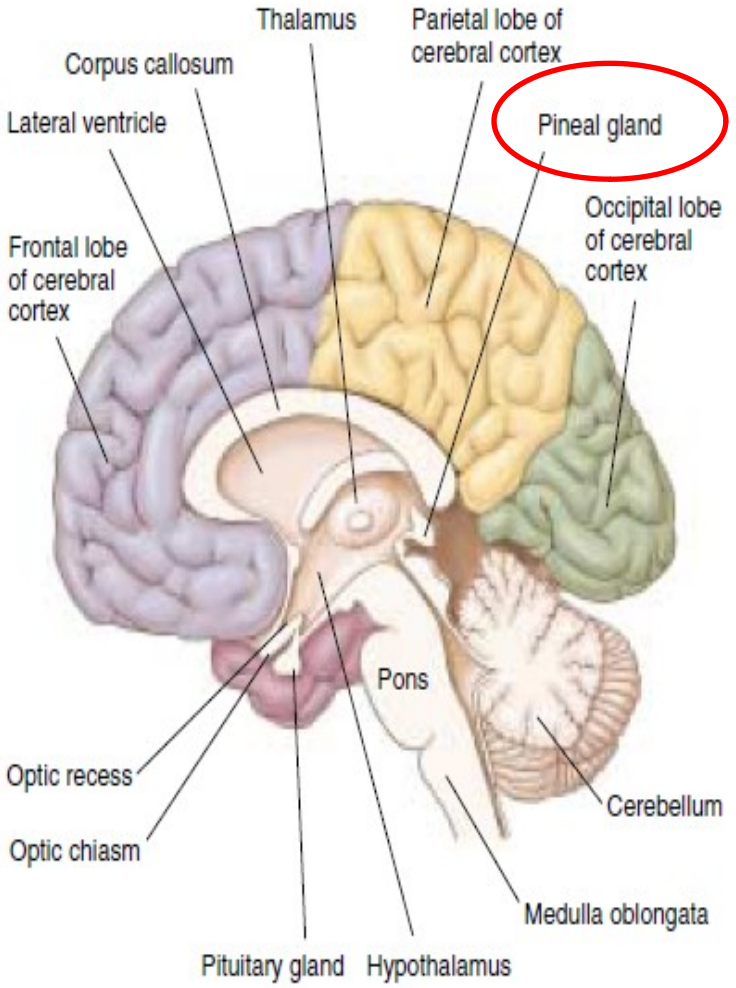
- **Pitressin**
- Increases **reabsorption** of water by **DCT** and collecting duct of nephron= reduces loss of water through urine (**Diuresis**)
- Also called **Antidiuretic hormone (ADH)**
- Absence of ADH → Urine output increases more than tenfold
- Drinking alcohol → Often causes frequent urination (alcohol inhibits secretion of ADH)
- High ADH → Also cause vasoconstriction → Increases blood pressure

Pineal gland

- **Epiphysis**
- Small endocrine gland
- **Location**: attached to the roof of the **3rd Ventricle Of Forebrain**
- Consist of masses of **Neuroglia** and **Secretory Cells** called **Pinealocytes**
- Secretes **Melatonin**

Melatonin

- Amine hormone
- Derived from **Serotonin** that itself is derived from the amino acid tryptophan
- Hormone of darkness
- Darkness of night = Secretion increases
- Light of day= secretion decreases
- Contribute to setting of the body's biological clock
- Potent antioxidant – provide protection against damaging



Source: Raven Johnson, Biology, 6th Edition, Page no.1093

Thyroid gland

- Largest endocrine gland in the body
- **Location**: on either side of the **trachea**
- Composed of right and left lateral lobes (one on either side of the trachea)= connected by a fibrous connective tissue – **Isthmus**
- Hormones secreted,

  Thyroid hormones (Thyroxine (T_4), Triiodothyronine (T_3))

  Calcitonin

Structure:

- Numerous spherical hollow sacs = **thyroid follicles and parafollicular cells**
- Lined with =simple cuboidal epithelium (composed of follicular cells
- Interior of follicles = **colloid**, protein- rich fluid
- **Follicular cells** produce,
  - **Thyroxine (T_4, Tetraiodothyronine)**
  - **Triiodothyronine (T_3)**

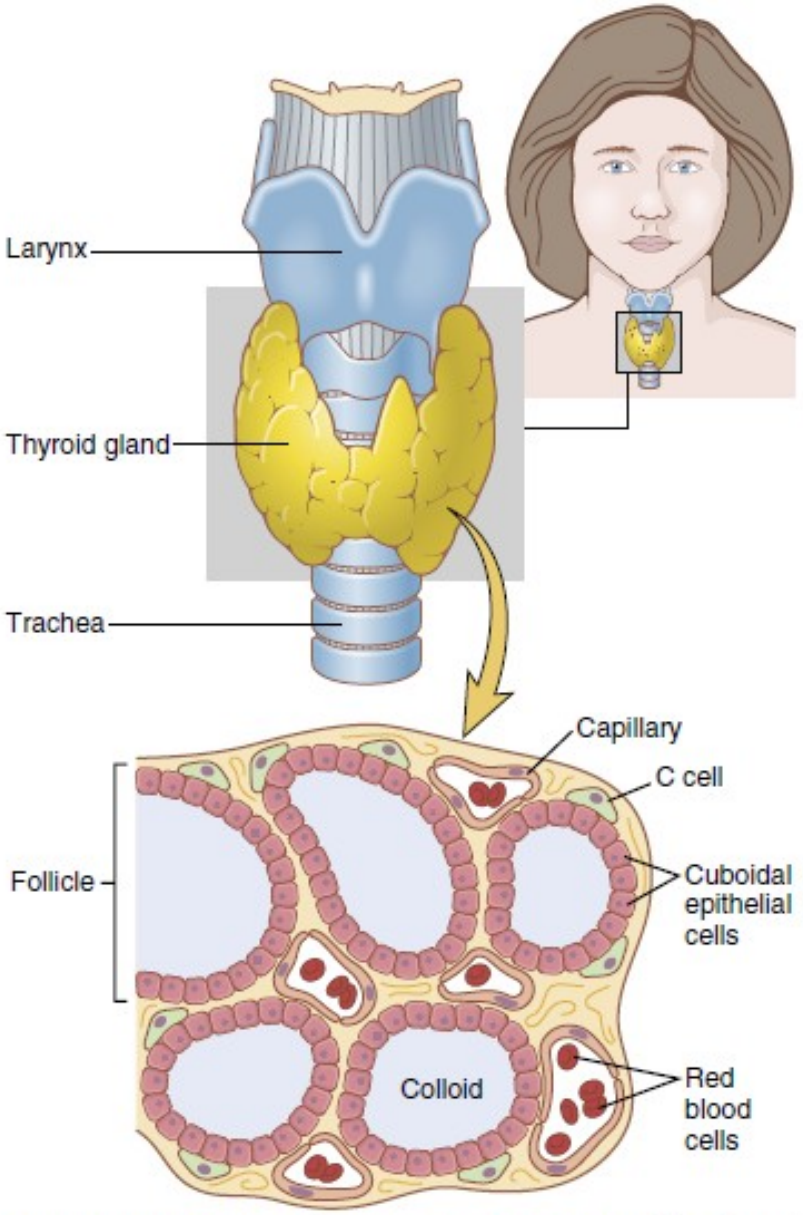


Figure 77-1 Anatomy and microscopic appearance of the thyroid gland, showing secretion of thyroglobulin into the follicles.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 892

Rashmi MG

oduction of Thyroid hormones

Follicular cells accumulate **iodide** from the blood and secrete it into the **colloid**

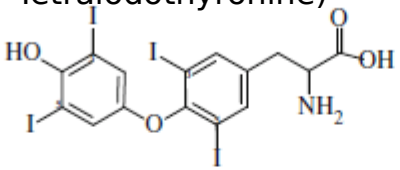
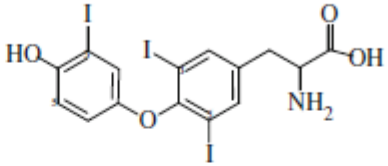
In the **colloid**,
Iodide ($2I^-$)→ **Iodine** (I_2) (oxidation)
Iodine (I_2) Attaches to a **tyrosine residue** of **thyroglobulin protein (secreted by follicular cells)**

1 Iodine + tyrosine residue of thyroglobulin = **Monoiodotyrosine (MIT)**
2 Iodine + tyrosine residue of thyroglobulin = **Diiodotyrosine (DIT)**

Inside the colloid,
Enzymes modify the structure of **MIT** and **DIT** = couples them together forming,
1 MIT+ 1 DIT = Triiodothyronine (T_3)
1 DIT+ 1 DIT = Thyroxine (T_4, Tetraiodothyronine)

Upon stimulation by **TSH (hypothalamus)**
Thyroid hormones which are Bound to thyroglobulin
Taken into the **follicular cells**

Hydrolysis reactions release free T_4 ,T_3

	Thyroxine (T_4, Tetraiodothyronine)	Triiodothyronine (T_3)
Structure:	 **78**	 **79**
Function:	Thyroxine and triiodothyronine are required in any cell for metabolism, for development, and for maturation.	
Receptor:	Two receptor subtypes with three (TR-α) and two (TR-β) splice variants; only TR-α_1, TR-β_1, and TR-β_2 can bind to the hormone; the receptors form homodimers, but preferentially form heterodimers with retinoid X receptor, a retinoic acid receptor.	

Source: Bernhard Kleine, Winfried G Rossmanith (2016), Hormones and the Endocrine system, Springer publications, page no. 240

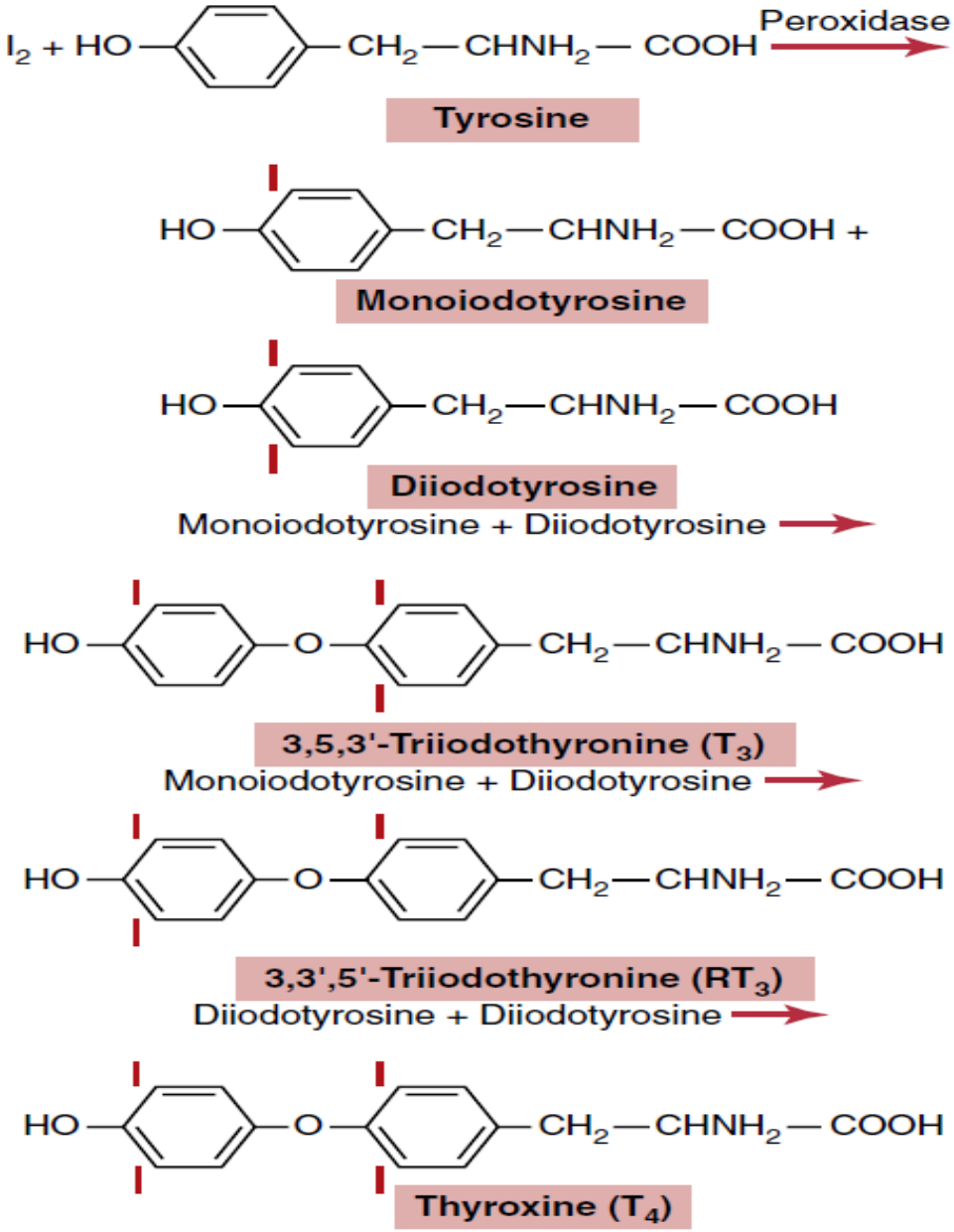

I2 + HO — CH2 — CHNH2 — COOH
Peroxidase
Tyrosine
HO — CH2 — CHNH2 — COOH +
Monoiodotyrosine
HO — CH2 — CHNH2 — COOH
Diiodotyrosine
Monoiodotyrosine + Diiodotyrosine
HO — O — CH2 — CHNH2 — COOH
3,5,3'-Triiodothyronine (T3)
Monoiodotyrosine + Diiodotyrosine
HO — O — CH2 — CHNH2 — COOH
3,3',5'-Triiodothyronine (RT3)
Diiodotyrosine + Diiodotyrosine
HO — O — CH2 — CHNH2 — COOH
Thyroxine (T4)

Figure 77-3 Chemistry of thyroxine and triiodothyronine formation.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 893

ormone secreted by thyroid gland = Thyroxine (T_4)

n the blood attached to **Carrier Proteins (Thyroxine-binding Globulin)**

also secretes a small amount of **T_3**

proteins have higher affinity for **T_4** than **T_3**

T_3 enter **target cells**

passes into the **target cell cytoplasm**

tically converted into **T_3**

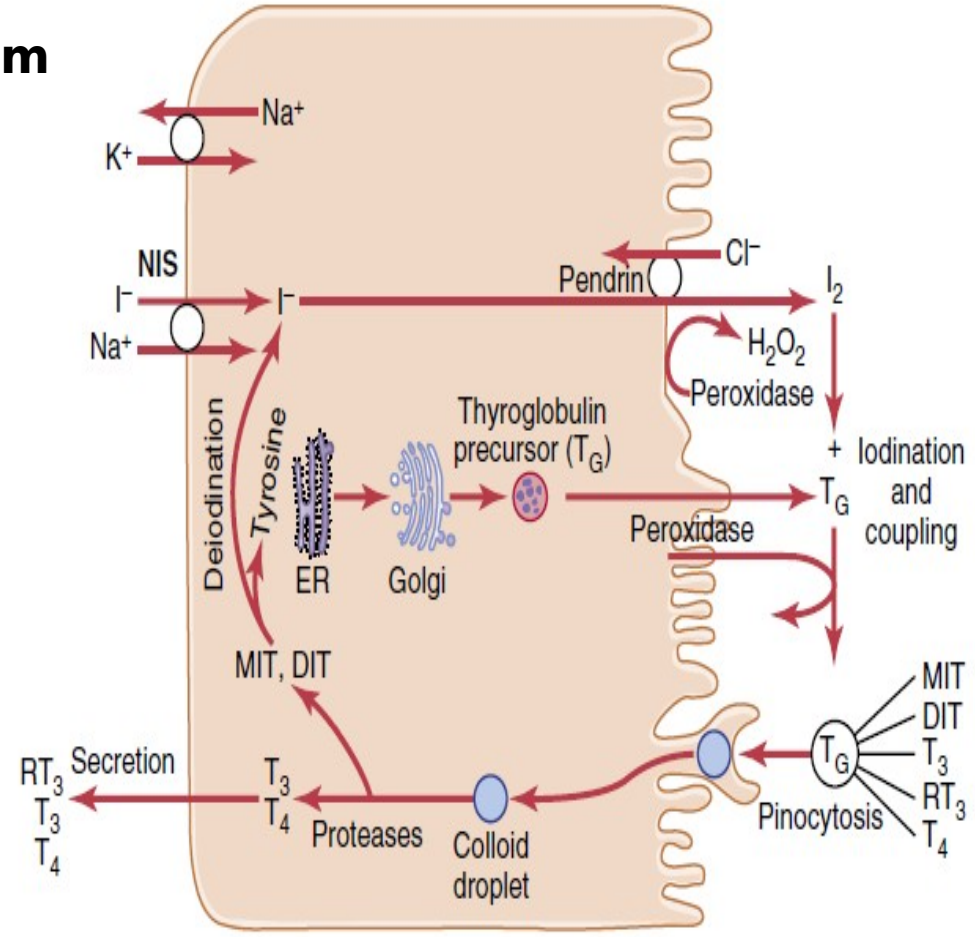


Figure 77-2 Thyroid cellular mechanisms for iodine transport, thyroxine and triiodothyronine formation, and thyroxine and triiodothyronine release into the blood. DIT, Diiodotyrosine; ER, endoplasmic reticulum; I^-, iodide ion; I_2, iodine; MIT, monoiodotyrosine; NIS, sodium-iodide symporter; RT_3, reverse triiodothyronine; T_3, triiodothyronine; T_4, thyroxine; T_G, thyroglobulin.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 892

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 895

Action of Thyroid Hormones

Mediated by their binding to nuclear receptors

Receptors of thyroid hormones = acts as **transcription factors** = regulate gene expression in target cells

Functions of Thyroid hormones

- Regulation of the **basal metabolic rate**
  By stimulating use of cellular oxygen = Increases basal metabolic rate in most tissues
- Stimulates synthesis of **Na^+ - K^+ ATPase**
- Together with **Human Growth Hormone** and **Insulin** = accelerates body growth, particularly the growth of the nervous and skeletal system

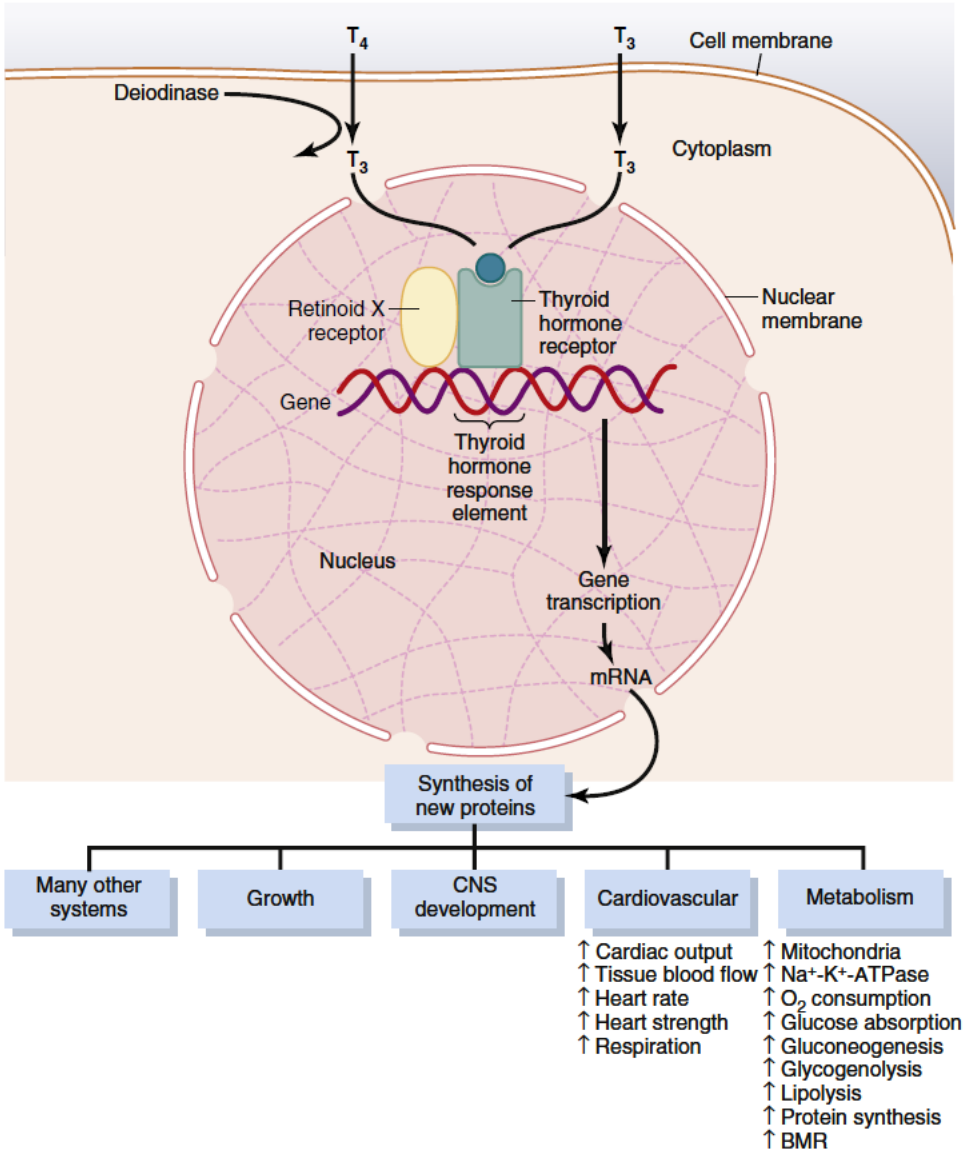


Figure 77-5 Thyroid hormone activation of target cells. Thyroxine (T_4) and triiodothyronine (T_3) enter the cell membrane by a carrier-mediated adenosine triphosphate–dependent transport process. Much of the T_4 is deiodinated to form T_3, which interacts with the thyroid hormone receptor, bound as a heterodimer with a retinoid X receptor, of the thyroid hormone response element of the gene. This action causes either increases or decreases in transcription of genes that lead to the formation of proteins, thus producing the thyroid hormone response of the cell. The actions of thyroid hormone on cells of several different systems are shown. BMR, Basal metabolic rate; CNS, central nervous system; mRNA, messenger ribonucleic acid; Na^+-K^+-ATPase, sodium–potassium–adenosine triphosphatase.

nin

e hormone

ed by **Parafollicular Cells** of **Thyroid Gland**

ce Calcitonin in response to **High Calcium Levels In Blood**

ases Mobilization Of Calcium from bones = Decrease plasma calcium concer

tes **Osteoblastic Activity**

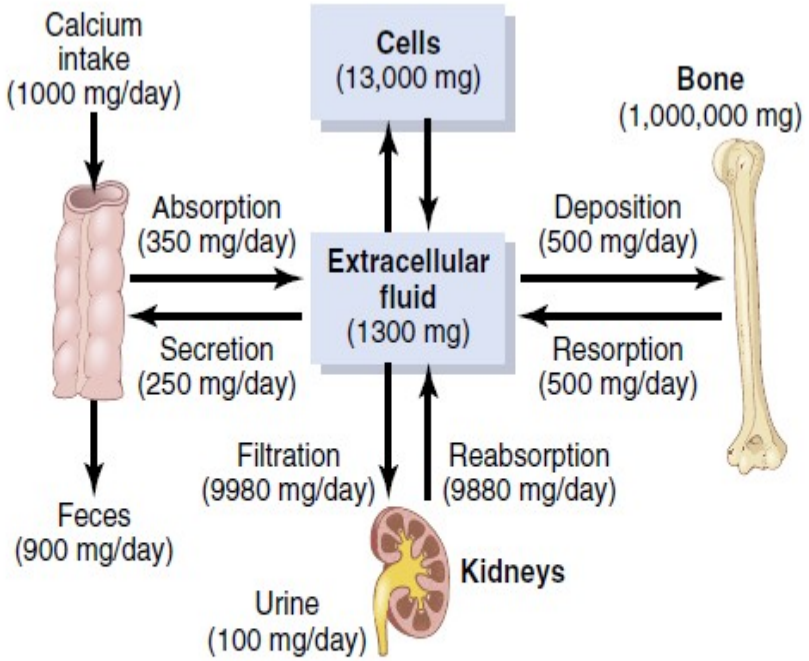


Figure 80-3. Overview of calcium exchange between different tissue compartments in a person ingesting 1000 mg of calcium per day. Note that most of the ingested calcium is normally eliminated in the feces, although the kidneys have the capacity to excrete large amounts by reducing tubular reabsorption of calcium.

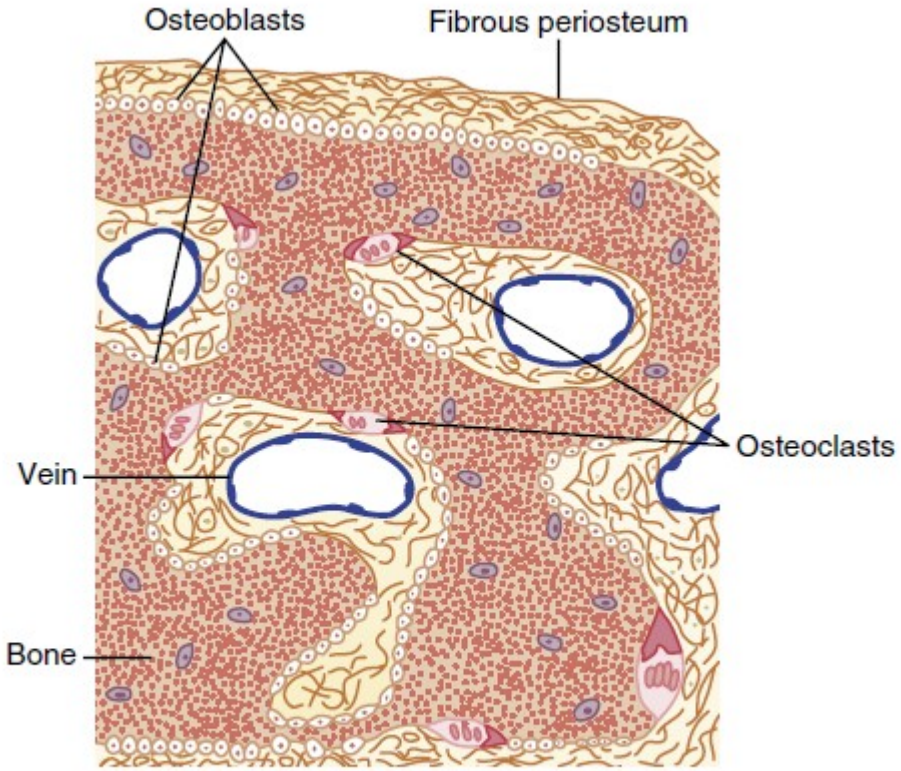


Figure 80-5. Osteoblastic and osteoclastic activity in the same bone.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 941-943

Parathyroid gland

- 4 small glands
- **Location**: backside of the thyroid gland (1 pair each in the 2 lobes of thyroid gland)
- **Hormones secreted**: Parathyroid hormone (PTH) or collip's hormone

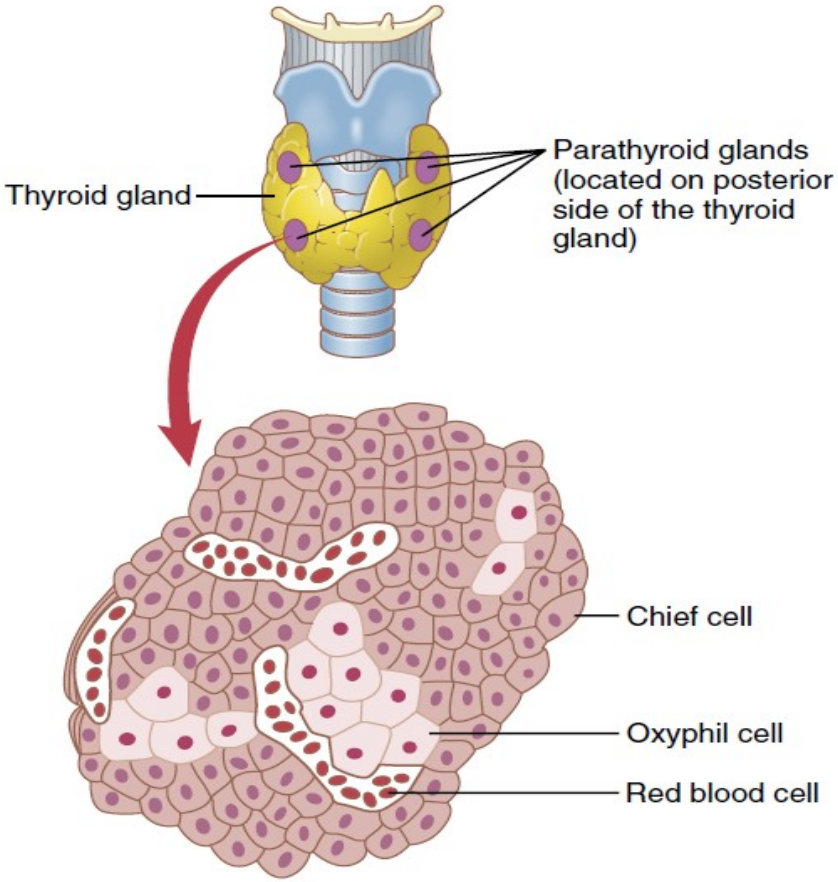


Figure 80-11. The four parathyroid glands lie immediately behind the thyroid gland. Almost all of the parathyroid hormone (PTH) is synthesized and secreted by the chief cells. The function of the oxyphil cells is uncertain, but they may be modified or depleted chief cells that no longer secrete PTH.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 948

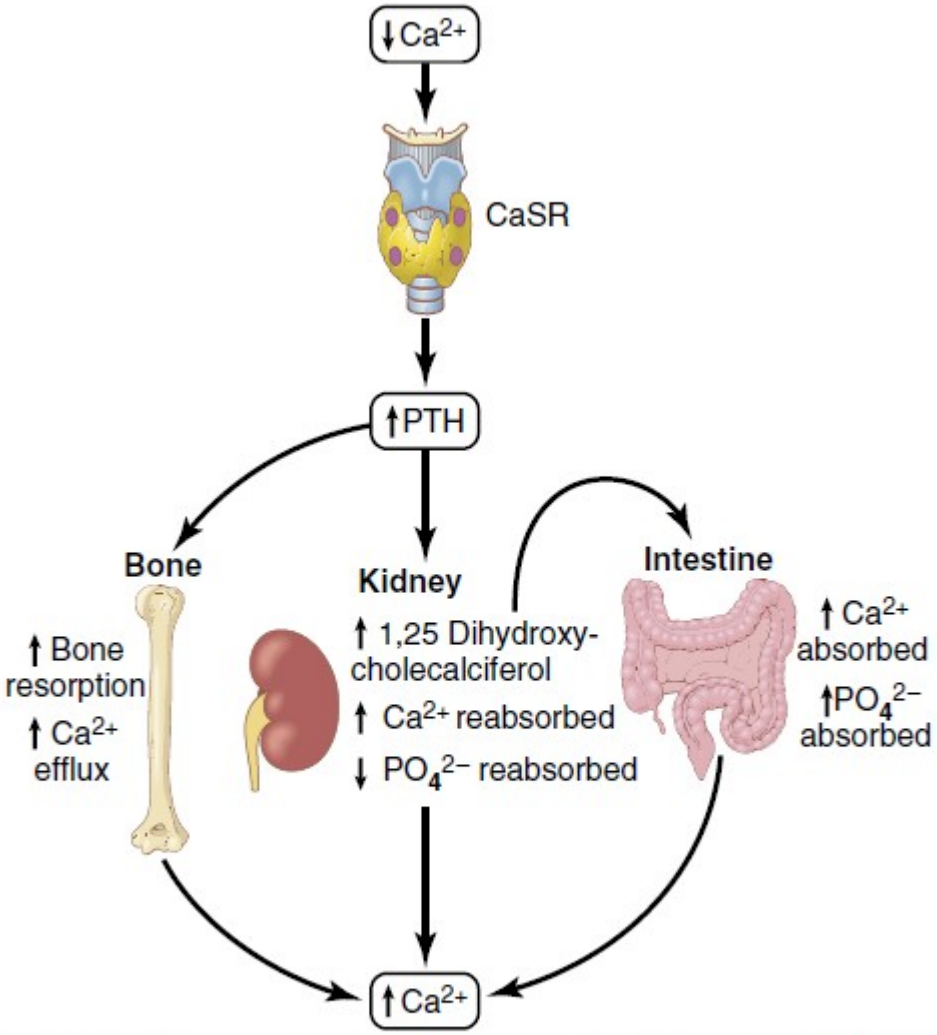


Figure 80-14. Summary of effects of parathyroid hormone (PTH) on bone, the kidneys, and the intestine in response to decreased extracellular fluid calcium ion concentration. CaSR, Calcium-sensing receptor.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th

Function of Parathyroid hormone

- Increases calcium in blood
- Secretion is regulated by calcium level in blood (not hypothalamic or pituitary hormones)
- Bone tissue= storage reservoir for calcium and PTH = stimulates removal of calcium from the bone = increases calcium level in blood = stimulates osteoclastic activity
- Increases reabsorption of calcium by renal tubules of kidney = less loss in urine = stimulates loss of phosphates in urine
- Stimulates kidney to secrete calcitriol = increases calcium absorption from the digested

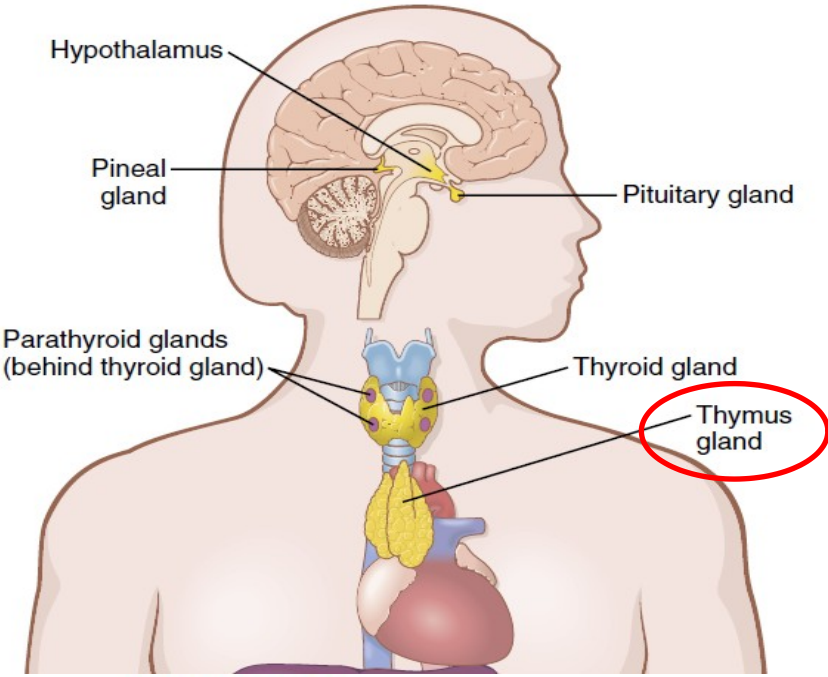
Rashmi MG

Thymus gland

- **Location**: behind the **sternum** between the **lungs**
- Grows during childhood, gradually decreases in size after puberty
- **Hormones secreted**: **Thymosin, Thymic Humoral Factor, Thymic Factor** and **Thymopoietin**

Functions of Thymus hormones

- Promote maturation of **T cells** (a type of WBC that destroys microbes and foreign substances)
- Retard the aging process
- **Thymus hormones** are called **Thymosin** = stimulate development and differentiation of T-lymphocytes / T-cells
- Role in regulating the immune system by stimulating other kinds of immune cells as well
- Responsible for growth during childhood



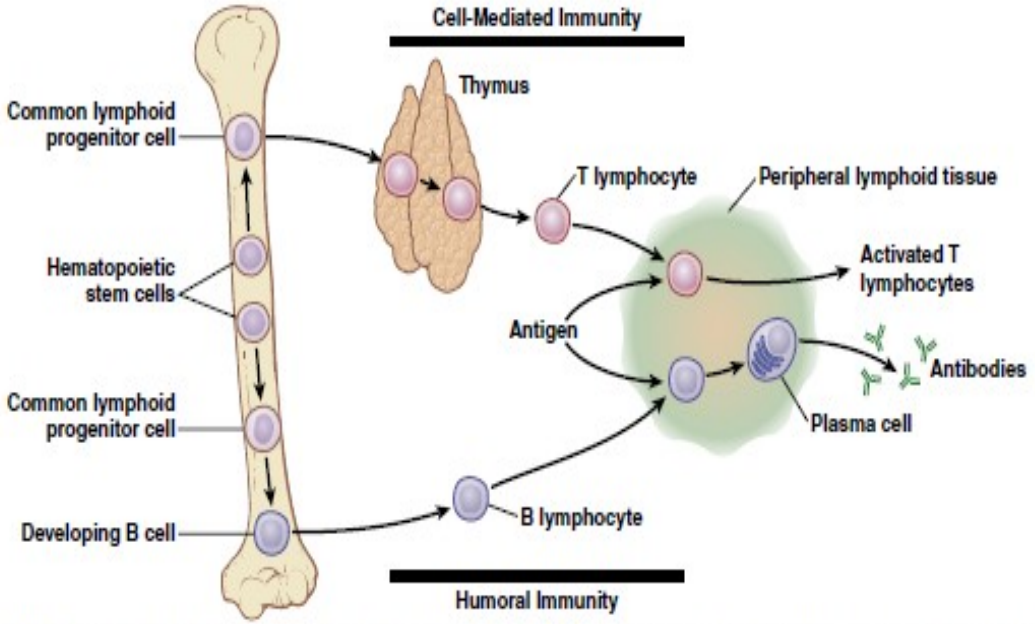


Figure 35-1. Formation of antibodies and sensitized lymphocytes by a lymph node in response to antigens. This figure also shows the origin of thymic (*T*) and bursal (*B*) lymphocytes that, respectively, are responsible for the cell-mediated and humoral immune processes.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 440,867

Pancreas

- **Composite gland** (acts as both Endocrine and Exocrine gland)
- Exocrine cells of pancreas = arranged in clusters = **Acini**

Structure:

- **Acini** = produce digestive enzymes = flow into GI tract = through pancreatic duct
- Scattered throughout the pancreas between Acini = **clusters / islands of endocrine cells = Islets of Langerhans (pancreatic islets)**
- Each pancreatic islets = include 4 types of hormone-secreting cells:
  - **Alpha or A- cells**
  - **Beta or B- cells**
  - **Delta or D-cells**
  - **F-cells**

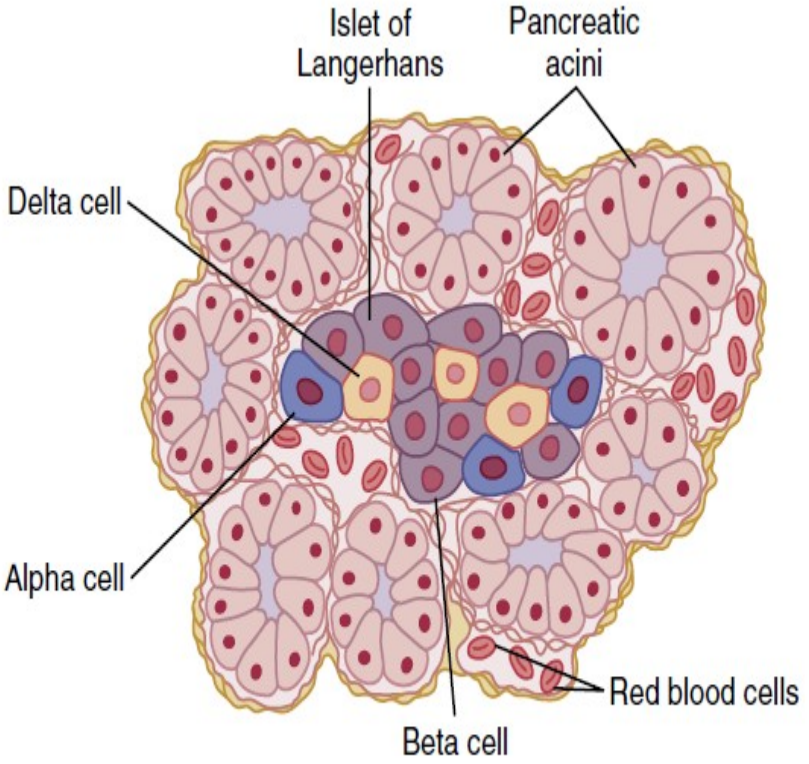


Figure 79-1. Physiological anatomy of an islet of Langerhans in the pancreas.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 922

- Alpha cells = 17% of pancreatic islet cells = secrete **Glucagon**
- Beta cells = 70% of pancreatic islet cells = secretes **Insulin**
- Delta cells = 7% pancreatic islet cells = secretes **Somatostatin** (inhibits the secretion of insulin and **Glucagon** = slows absorption of nutrients from GI tract)
- F cells = secretes **Pancreatic Polypeptide** (inhibits Somatostatin

Glucagon

- Linear polypeptide of 29 amino acid resides
- Stimulates **Glycogenolysis** = **Increases** level of glucose in blood (**Hyperglycemia**)
- Also stimulates the process of **Gluconeogenesis**

Functions:

- **Glycogenolysis** (breakdown of **Glycogen** into **Glucose** = **Increase** level of **Glucose**
- **Glycogenesis** (production of **Glycogen** from **Glucose**)= **Decrease** level of **Glucose**
- **Gluconeogenesis** (synthesis of **Glucose** from **Non-carbohydrate**) **= Increase** level of **Glucose**
- **Triacylglycerol Hydrolysis** (breaking of **Fats** using water to yield **1 Glycerol** = **Glycerol** converted into **Glucose** via **Gluconeogenesis**) = **Increase** level of **Glucose**
- Blood glucose level
- **Glycolysis** (breakdown of **Glucose** into **Pyruvate**) = **Decrease** level of **Glucose**
- **Glucose** release from liver = **Increase** level of **Glucose**

Rashmi MG

Insulin

- Protein made up of 2 polypeptide chains (A- 21 amino acid residues) and (B- 30 amino acid residues) joined by 2 pairs of disulfide bonds with an additional intramolecular **Disulfide Bond** in the **A Chain**

Synthesis

- First synthesized as **Preproinsulin** (110 amino acid residues) = **Inactive Precursor**
- Processing **Preproinsulin** = removal of signal peptide from **N- Terminus** of **Preproinsulin** = generates **Proinsulin** molecule
- **Proteolytic Cleavage** = removes **C-peptide Chain** = produces **Mature Insulin**

Functions

- Accelerates transport of glucose into cells = Lowers blood glucose level
- Stimulates **Glycogenesis** in both skeletal muscle and the liver
- Inhibits **Glycogenolysis**
- Inhibits **Gluconeogenesis** in liver
- Enhances entry of fatty acids from blood into adipose tissue cells = increases **Lipogenesis** = inhibits **Lipolysis**

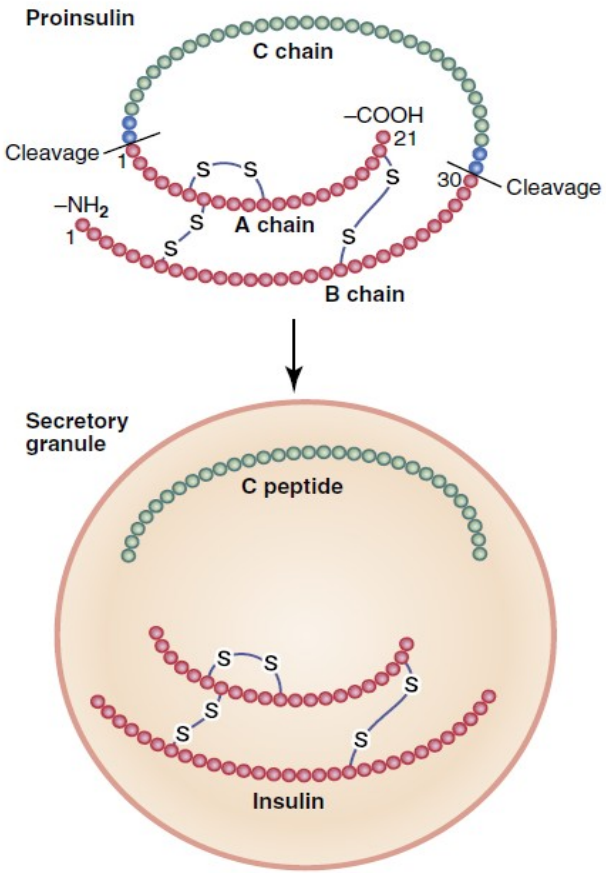


Figure 79-2. A schematic of the human proinsulin molecule, which is cleaved in the Golgi apparatus of the pancreatic beta cells to form connecting peptide (C peptide), and insulin, which is composed of the A and B chains connected by disulfide bonds. The C peptide and insulin are packaged in granules and secreted in equimolar amounts, along with a small amount of proinsulin.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 923

Mechanism of glucose uptake by cell in the presence of Insulin

- Insulin = lowers glucose level = by accelerating transport of glucose into cells
- Glucose transport (between blood and cells) = plasma membrane bound carrier protein = **Glucose Transporter (GLUT)**
- **GLUT-4** = transporter that responds to insulin (usually not present in plasma membrane in the absence of Insulin)
- Stored in intracellular vesicles, Upon **Insulin** stimulation = fuses with plasma membrane = increasing the rate of sugar uptake by several-fold

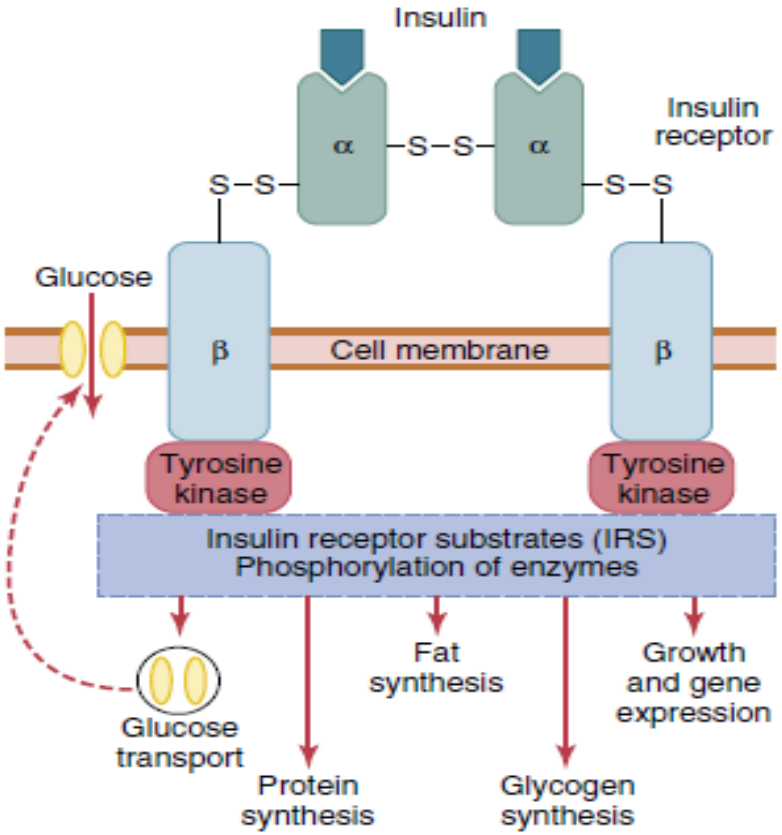


Figure 79-3. A schematic of the insulin receptor. Insulin binds to the α subunit of its receptor, which causes autophosphorylation of the β-subunit receptor, which in turn induces tyrosine kinase activity. The receptor tyrosine kinase activity begins a cascade of cell phosphorylation that increases or decreases the activity of enzymes, including insulin receptor substrates, that mediate the effects on glucose, fat, and protein metabolism. For example, glucose transporters are moved to the cell membrane to assist glucose entry into the cell.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 924

Rashmi MG

gulation of Glucagon and insulin secretion

Low blood glucose (**Hypoglycemia**)

↓

Stimulates **Alpha Cells** to secrete **Glucagon**

↓

Glucagon acts on Hepatocytes to induce,
- **Gluconeogenesis** (synthesis of glucose from non-carbohydrate) = Increase level of glucose
- **Glycogenolysis** (breakdown of glycogen into glucose = increase level of glucose

↓

Glucose released by **Hepatocytes** raises blood glucose level to normal

↓

If blood glucose continues to rise = **Hyperglycemia Inhibits** release of **Glucagon**

High blood glucose (**Hyperglycemia**

↓

Stimulates **Beta Cells** to secrete **Insulin**

↓

Insulin accelerates,
- **Diffusion** of glucose into cells
- Increases **Glycogenesis** and **Lipogenesis**
- Slows **Glycogenolysis** and **Gluconeogenesis**

↓

Blood glucose level falls

↓

If blood glucose continues to fall, **Hypoglycemia Inhibits** release of **Insulin**

enal glands

r of adrenal gland

tion : on each **kidney** (located superior to kidney = **suprarenal glands**)

mones secreted: Steroid hormones, **Epinephrine**, **Norepinephrine**

ture:

ed into 2 regions:

e peripherally located = **Adrenal Cortex** (80-90%)

l centrally located = **Adrenal Medulla** (10%)

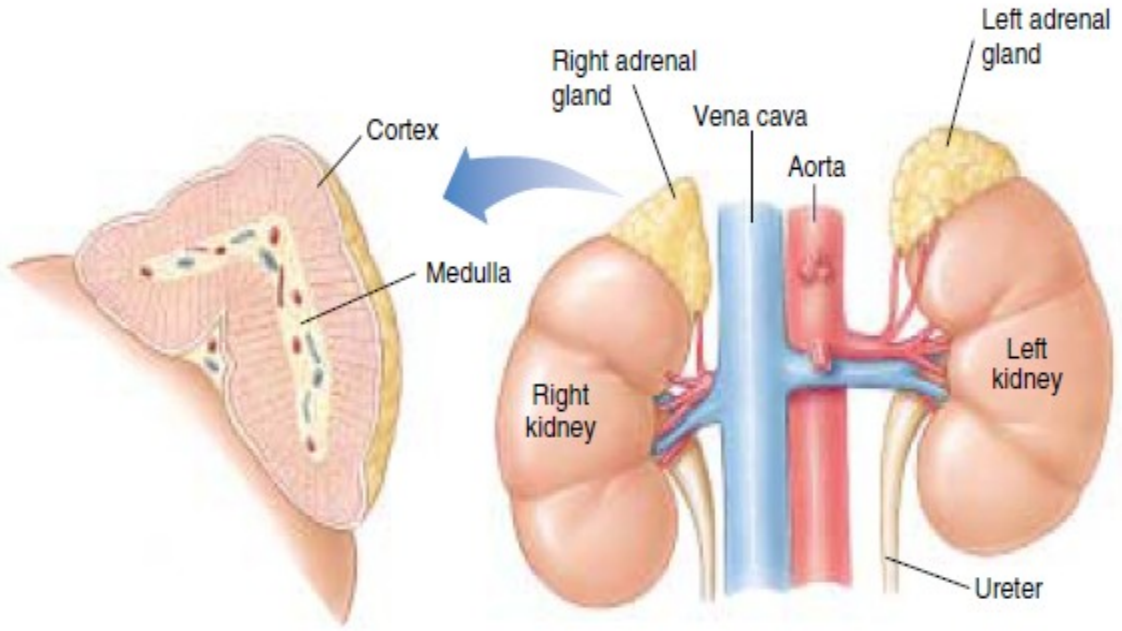


FIGURE 56.19
The adrenal glands. The inner portion of the gland, the adrenal medulla, produces epinephrine and norepinephrine, which initiate a response to stress. The outer portion of the gland, the adrenal cortex, produces steroid hormones that influence blood glucose levels.

Source: Raven Johnson, Biology, 6th Edition, Page no.1143

al cortex

ded into 3 regions (Each secretes different hormones):

glomerulosa = outermost layer = Secretes **Mineralocorticoids**

asciculata = middle and largest portion = Secretes Glucocorticoids

eticularis = Innermost zone = Secretes **sex hormones (Adrenal androgens**

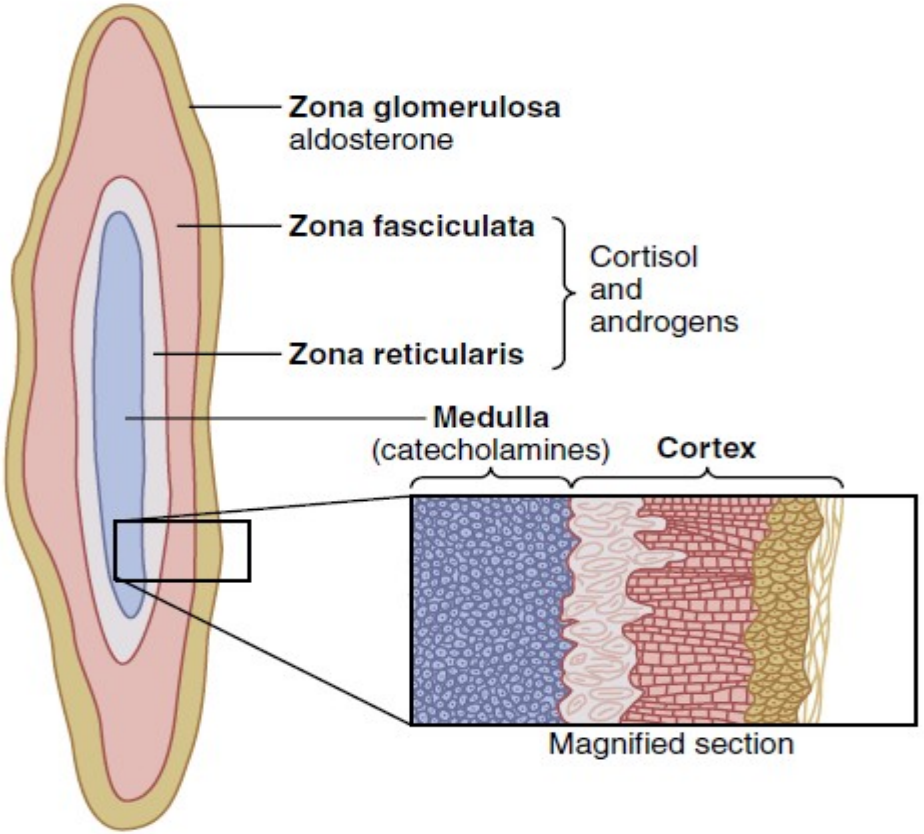


Figure 78-1. Secretion of adrenocortical hormones by the different zones of the adrenal cortex and secretion of catecholamines by the adrenal medulla.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 904

osterone

jor **Mineralocorticoids** from **Zona glomerulosa**

ctions:

gulates homeostasis of 2 mineral ions (**Na^+ ions** and **K^+ ions**)

intenance of electrolytes, body fluid volume, osmotic pressure and blood pressu

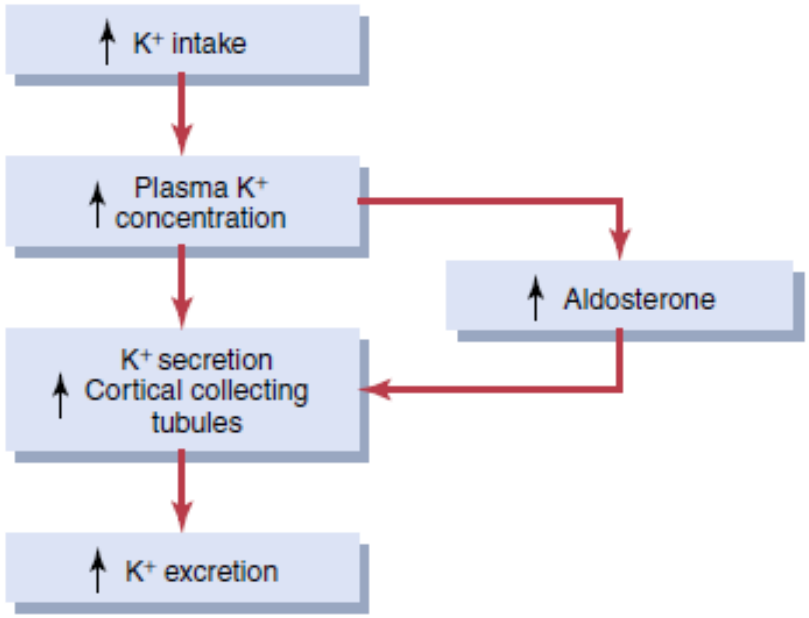


Figure 30-7. Primary mechanisms whereby high potassium intake raises potassium excretion. Note that increased plasma potassium concentration directly raises potassium secretion by the cortical collecting tubules and indirectly increases potassium secretion by raising plasma aldosterone concentration.

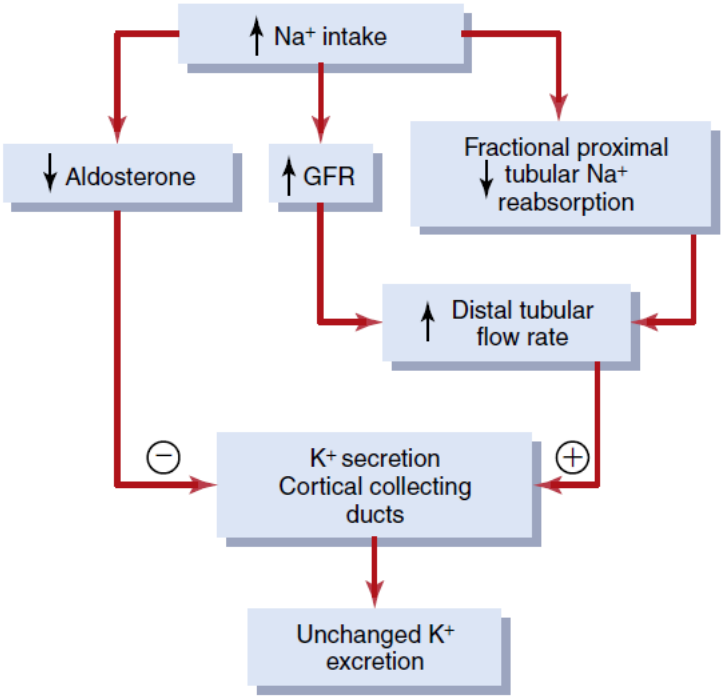


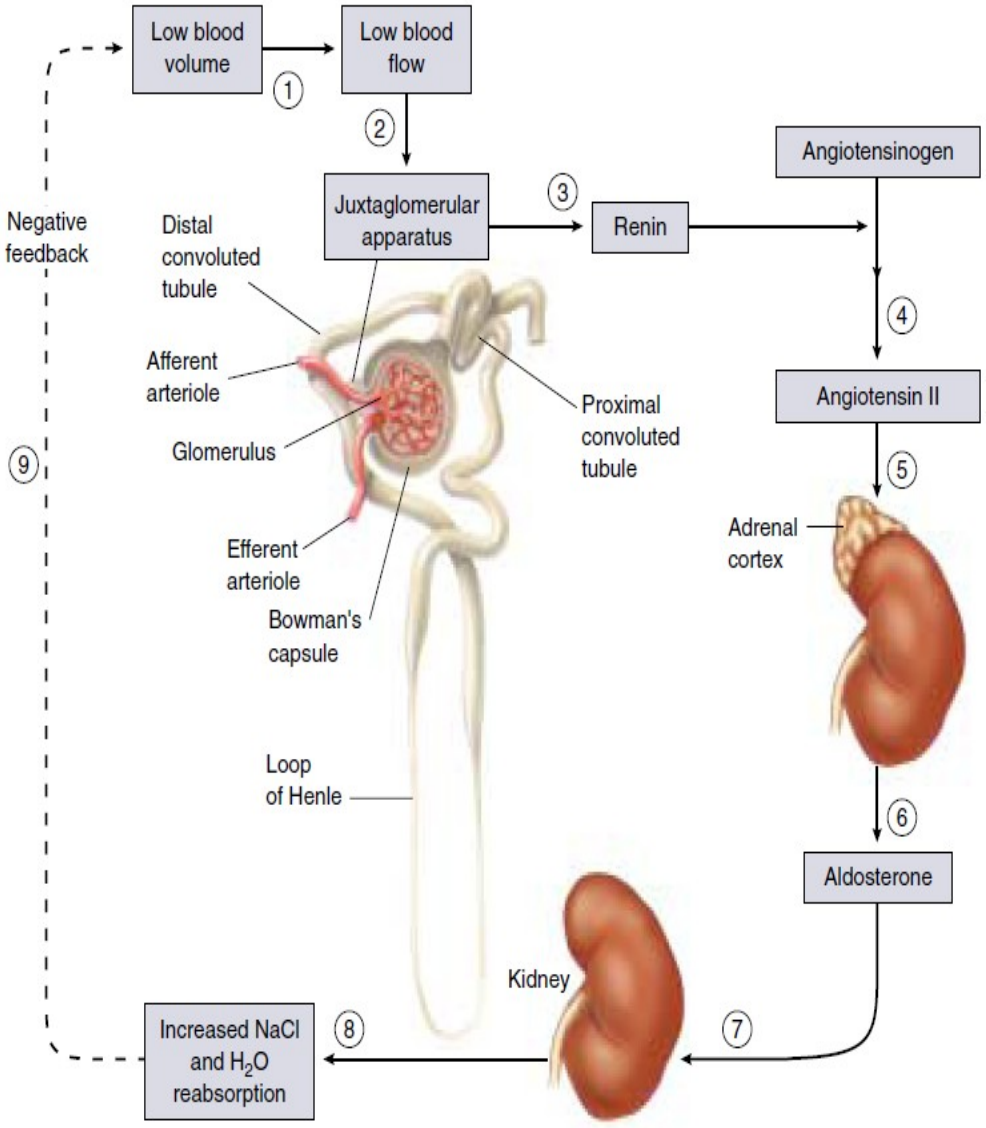
Figure 30-10. Effect of high sodium intake on renal excretion of potassium. Note that a high-sodium diet decreases plasma aldosterone, which tends to decrease potassium secretion by the cortical collecting tubules. However, the high-sodium diet simultaneously increases fluid delivery to the cortical collecting duct, which tends to increase potassium secretion. The opposing effects of a high-sodium diet counterbalance each other, so there is little change in potassium excretion. GFR, Glomerular filtration rate.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no. 373,374

Renin-angiotensin-aldosterone system

- Controls secretion of **aldosterone**
- Acts on kidney to promote absorption of **sodium** and excretion of **potassium**
- In **presence** of **aldosterone**, Increased level of Na^+ ions = **retention of water = increased blood volume**
- In **absence** of **aldosterone**,

FIGURE 58.24
A lowering of blood volume activates the renin-angiotensin-aldosterone system. (*1*) Low blood volume accompanies a decrease in blood Na^+ levels. (*2*) Reduced blood flow past the juxtaglomerular apparatus triggers (*3*) the release of renin into the blood, which catalyzes the production of angiotensin I from angiotensinogen. (*4*) Angiotensin I converts into an active form, angiotensin II. (*5*) Angiotensin II stimulates blood vessel constriction and (*6*) the release of aldosterone from the adrenal cortex. (*7*) Aldosterone stimulates the reabsorption of Na^+ in the distal convoluted tubules. (*8*) Increased Na^+ reabsorption is followed by the reabsorption of Cl^- and water. (*9*) This increases blood volume. An increase in blood volume may also trigger the release of atrial natriuretic hormone that inhibits the release of aldosterone. These two systems work together to maintain homeostasis.



Source: Raven Johnson, Biology, 6th Edition, Page no.1194

Rashmi MG

Glucocorticoids from Zona fasciculata

- Involved in carbohydrate metabolism
- Include **Cortisol** (**Hydrocortisone**), **Corticosterone** and **Cortisone** (Principal Glucocorticoids = **Cortisol**)
- Secretion is stimulated by **ACTH** from anterior pituitary
- **ACTH** promotes secretion of **cortisol**

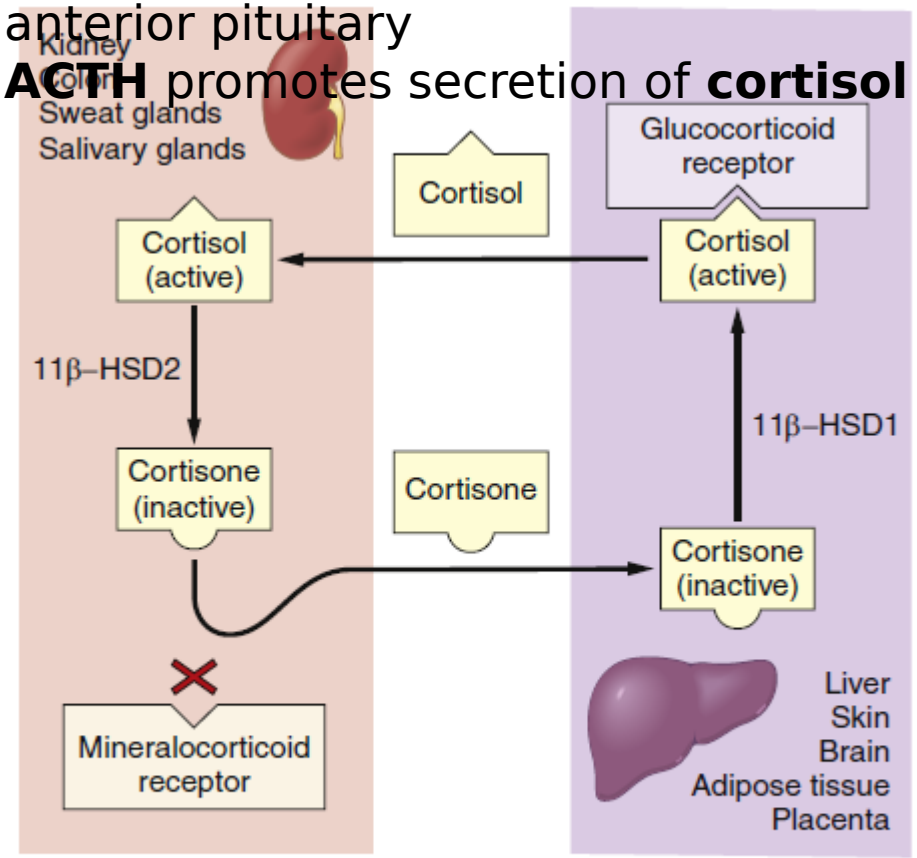


Figure 78-7. Interconversion of active cortisol and inactive cortisone by the two 11β-hydroxysteroid dehydrogenase isoforms (11β-HSD1 and 11β-HSD2) in various tissues.

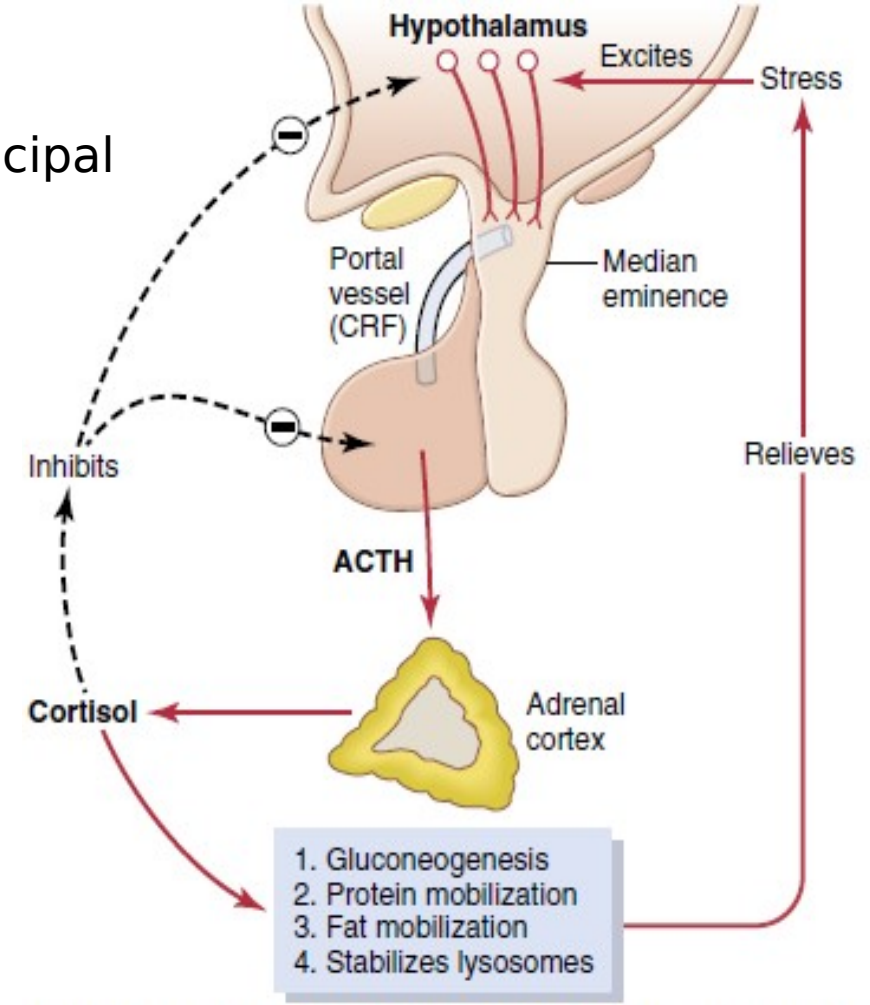


Figure 78-8. Mechanism for regulation of glucocorticoid secretion. ACTH, Adrenocorticotropic hormone; CRF, corticotropin-releasing factor.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no.915, 916

Effects of Glucocorticoids

- (↑) = rate of protein breakdown in muscle fibers = (↑) liberation of amino acids into bloodstream
- Induces gluconeogenesis in liver
- Stimulates Lipolysis, breakdown of triglycerides = release fatty acids from adipose tissue to blood
- Provide resistance to stresses (exercise, fasting, fright, bleeding, infection and disease)
- Inhibits WBC that participate in inflammatory responses
- retard tissue repair = slow wound healing
- (↑) = doses of Glucocorticoids = depress immune responses (used as immunosuppressive agent = prescribed for organ transplant recipients to retard tissue rejection by the immune system

Adrenal androgens from Zona reticularis

- Principal adrenal androgen = **DHEA** = **DeHydroEpiAndrosterone**
- Belongs to class of hormones known as androgens
- Stimulates growth of axial hairs, facial hairs and pubic hairs in boys and girls
- Includes major male sex hormones = **Testosterone** = produced by Testes
- In females = promotes libido (sex drive) = converted into estrogen by other body tissue
- After menopause,
- Ovarian secretion of

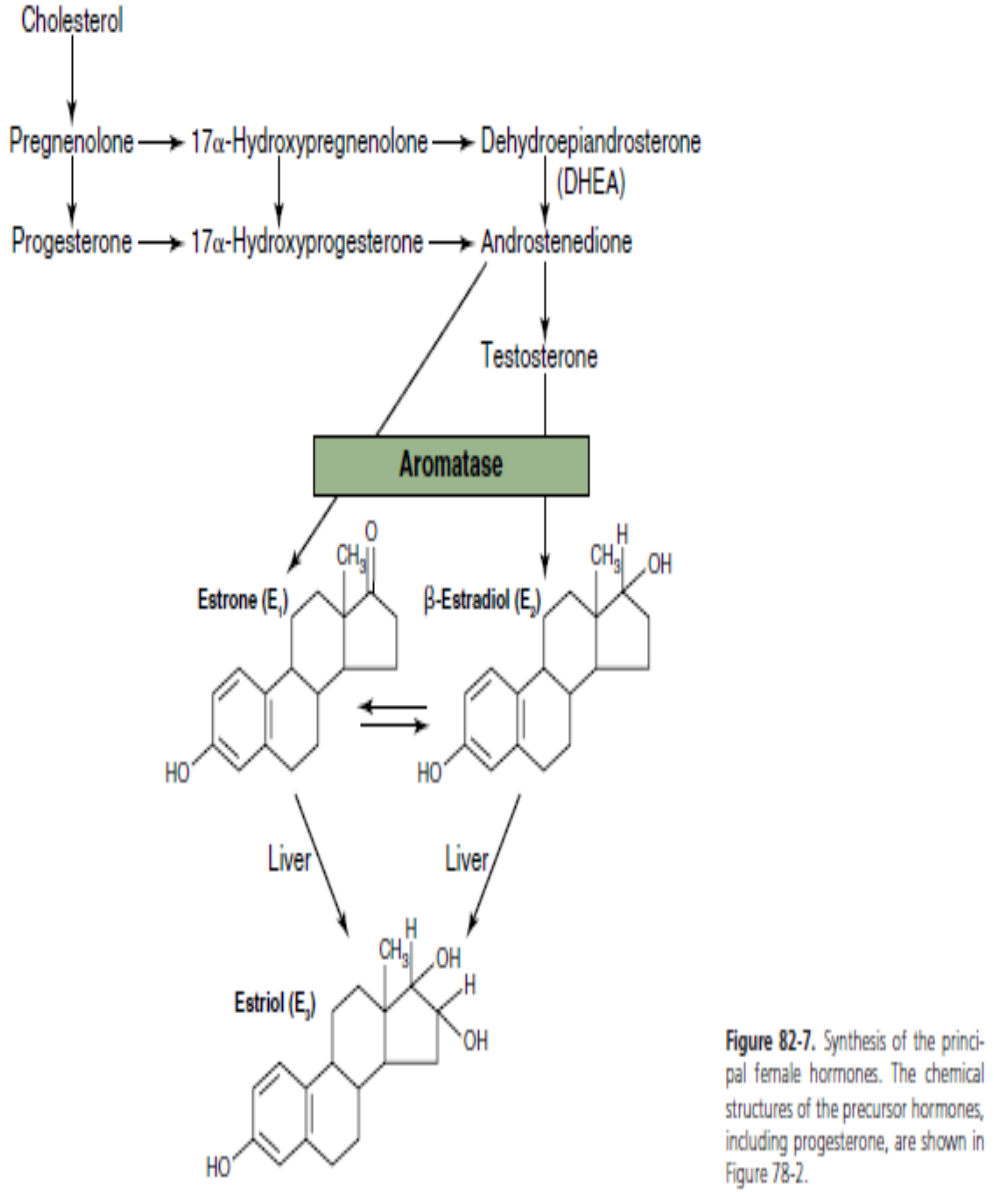


Figure 82-7. Synthesis of the principal female hormones. The chemical structures of the precursor hormones, including progesterone, are shown in Figure 78-2.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier

Adrenal medulla

- Modified part of **Sympathetic Nervous System**
- Consists of modified **postganglionic sympathetic neurons** (do not have axons)
- Release hormones directly into the blood rather than releasing a neurotransmitter
- Hormone producing cells = **Chromaffin cells**
- **Hormones secreted: Epinephrine(adrenaline**)= 80% and **Norepinephrine** (noradrenaline) = 20% (collectively called **Catecholamines**)
- Both hormones are synthesized from amino acid **tyrosine**

Functions:

- Emergency / stress condition (injury, pain, fear, accident an grief) →Impulse from **Hypothalamus** stimulate **sympathetic preganglionic neurons** → Stimulates **Chromaffin cells** to secrete **Epinephrine**, **Norepinephrine** = **Emergency hormones / Fight / flight**
- Increases heart rate and force of contraction → the output of heart
- Increases systolic blood pressure
- Increase blood flow to the heart , liver, skeletal muscles and adipose tissue

Gonadal Hormones

- Primary reproductive organs = **Gonads**
- Produce gametes
- In males = Testes = produce Sperms
- In females = Ovaries = produce oocytes
- Secretes Hormones

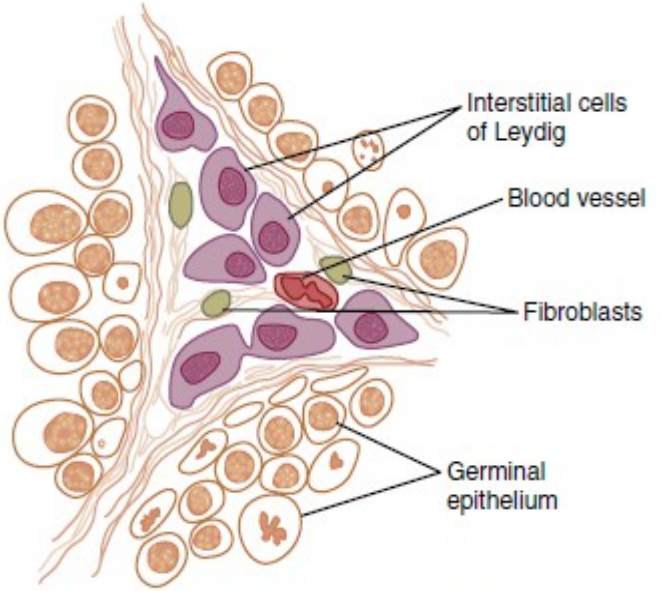


Figure 81-7 Interstitial cells of Leydig, the cells that secrete testosterone, located in the interstices between the seminiferous tubules.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no.965

Endocrine part of Testes = Interstitial cells or Leydig's cells

- **Hormones secreted: Androgen** (male sex hormone)
- Principal androgen = **Testosterone**

Functions:

- Stimulates spermatogenesis
- Responsible for growth and development of male accessory sex organs (epididymis, vas deferens, seminal vesicles, prostrate gland, urethra etc.)
- Stimulates muscular growth, growth of facial and axillary hairs
- Causes aggressiveness, low pitch of voice
- Influences male sexual behavior (libido)

Rashmi MG

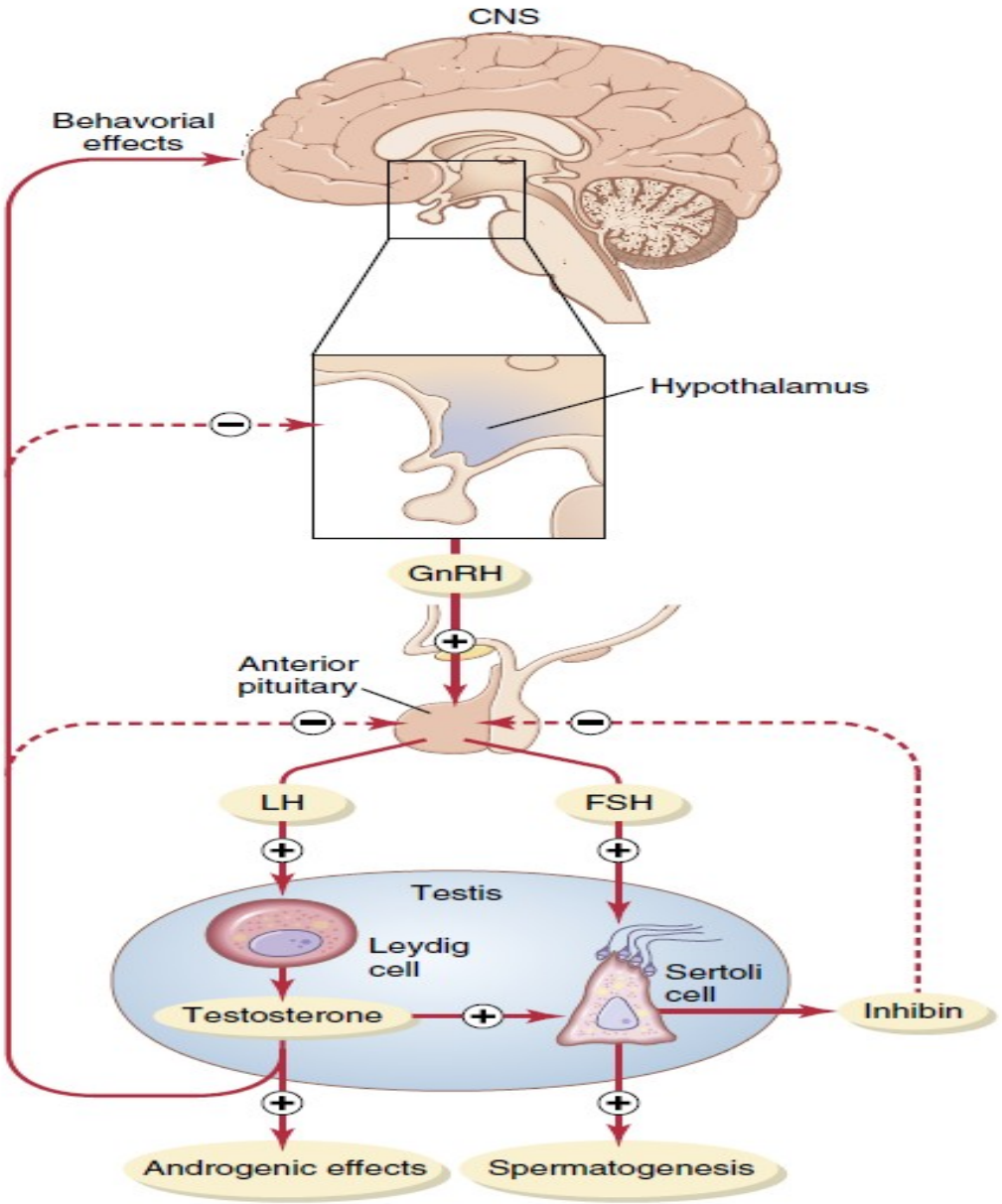


Figure 81-10 Feedback regulation of the hypothalamic-pituitary-testicular axis in males. Stimulatory effects are shown by *plus signs*, and negative feedback inhibitory effects are shown by *minus signs*. CNS, Central nervous system; FSH, follicle-stimulating hormone; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no.969

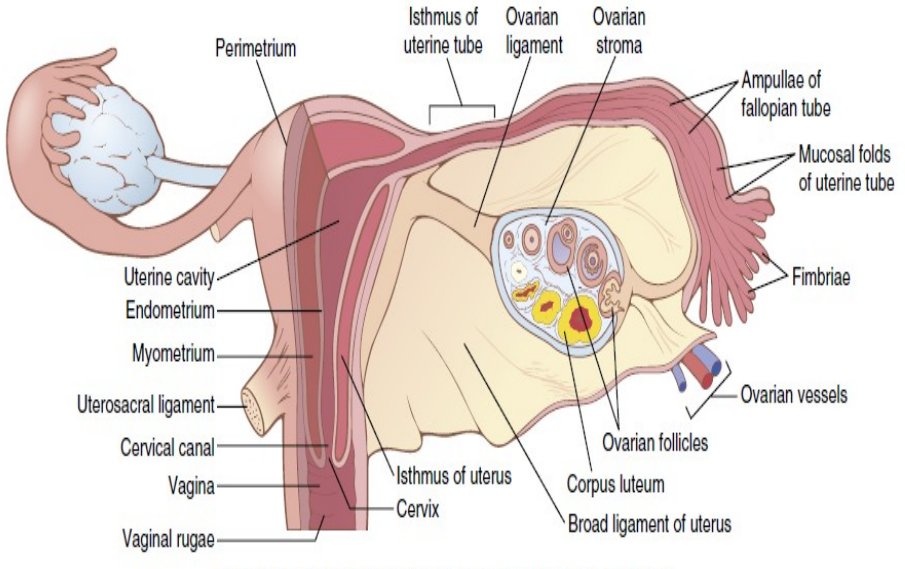


Figure 82-2. Internal structures of the uterus, ovary, and a uterine tube.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no.974,975

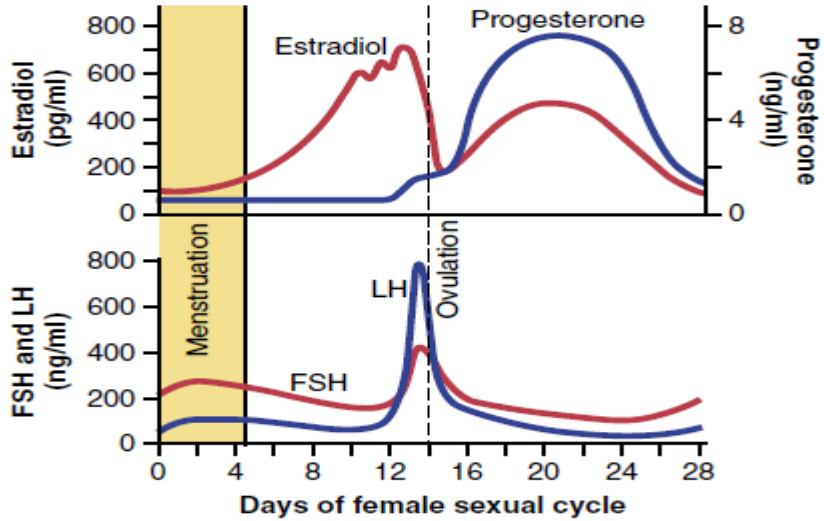


Figure 82-4. Approximate plasma concentrations of the gonadotropins and ovarian hormones during the normal female sexual cycle. FSH, Follicle-stimulating hormone; LH, luteinizing hormone.

Endocrine part of ovaries

- **Hormones secreted: Estrogens** (**Estradiol** and **Estrone**), **Progesterone**, **Inhibin** and **Relaxin**

Functions:

- Regulates menstrual cycle, maintain pregnancy, prepare mammary glands for lactation, promote enlargement of breast
- Helps maintain female secondary sex characteristics
- **Estrogen** = secreted by **Graafian follicle (mature follicle)**
- Stimulated by **LH** and **FSH** (of pituitary gland)
- **Progesterone** = secreted by **Corpus luteum**
- (small amount is secreted by adrenal cortex and placenta)
- Responsible for maintenance of pregnancy = **pregnancy hormone**
- Inhibin = inhibits secretion of **FSH**
- **Relaxin** = secreted during

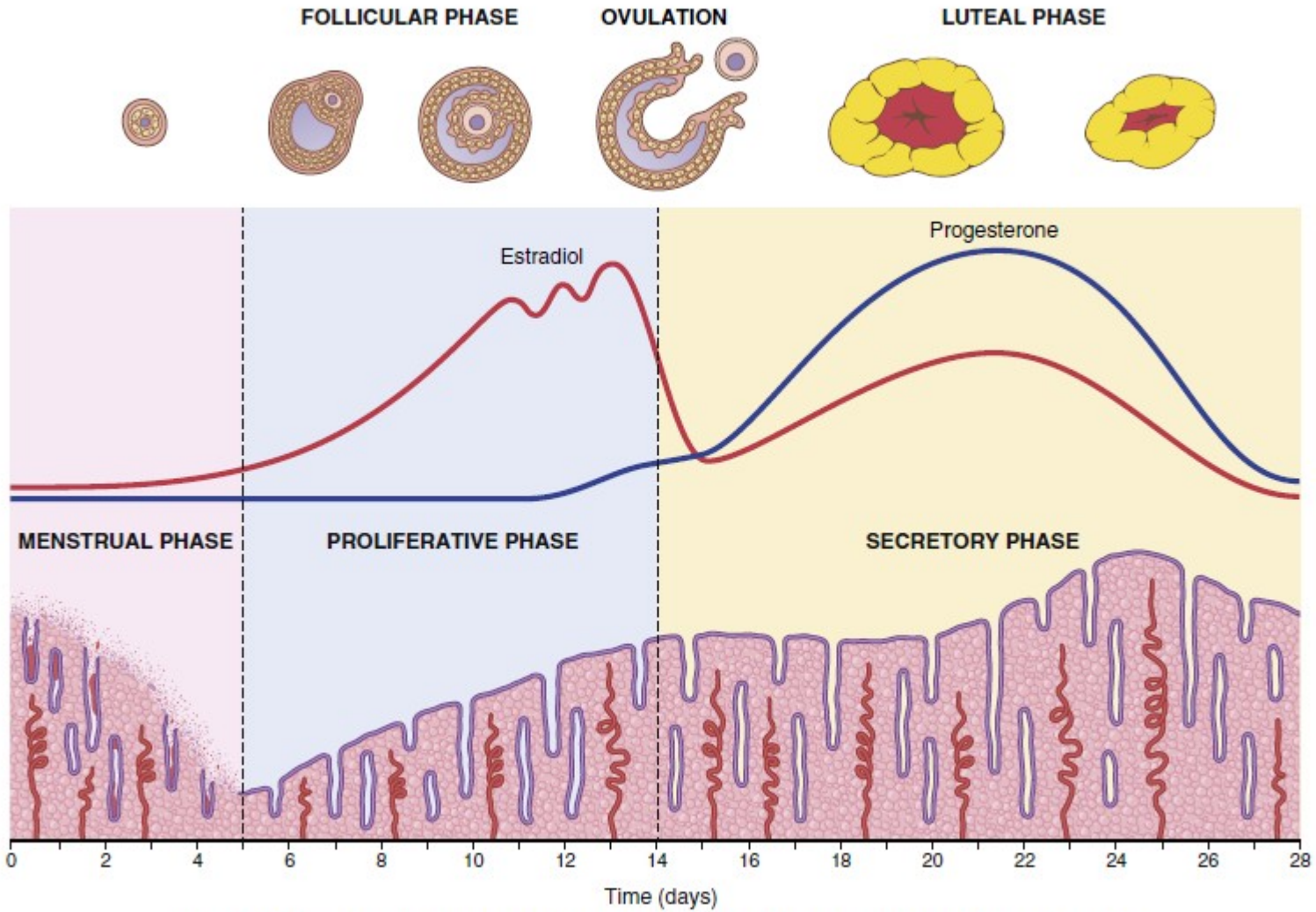


Figure 82-9. Phases of endometrial growth and menstruation during each monthly female sexual cycle.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier

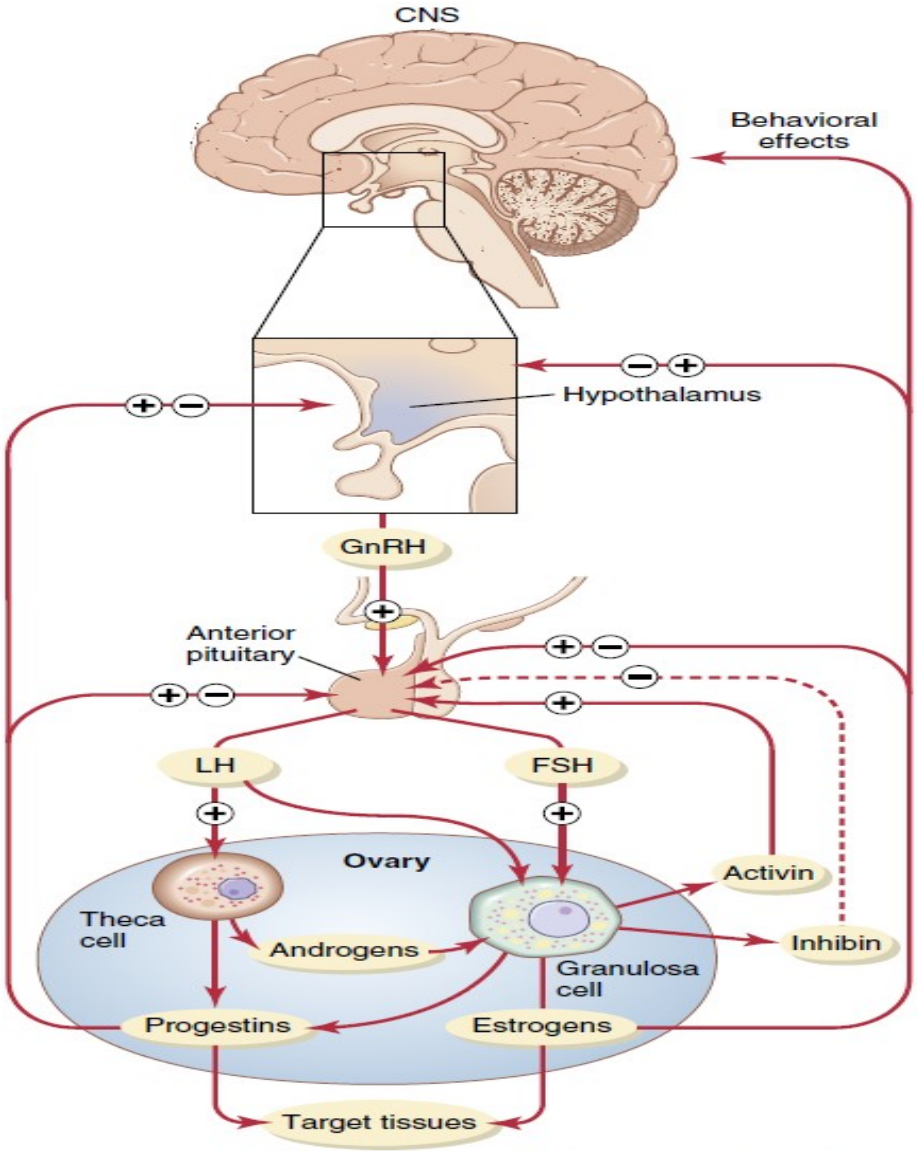


Figure 82-11. Feedback regulation of the hypothalamic-pituitary-ovarian axis in females. Stimulatory effects are shown by plus signs (+); negative feedback inhibitory effects are shown by minus signs (–). Estrogens and progestins exert negative and positive feedback effects on the anterior pituitary and hypothalamus, depending on the stage of the ovarian cycle. Inhibin has a negative feedback effect on the anterior pituitary, whereas activin has the opposite effect, stimulating follicle-stimulating hormone (FSH) secretion by the anterior pituitary. CNS, Central nervous system; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone.

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no.985

rmones from kidney, heart, placenta and GI tract

1. From kidney

Location	Hormones secreted
Juxtaglomerular cells	**Renin**
• **Renin** initiates **Renin-Angiotensin-Aldosterone system** • Catalyzes conversion of **Angiotensin** (Released by Liver) → **Angiotensin I** • **Angiotensin I** hydrolyzed by **Angiotensin converting enzymes** → forms **Angiotensin II** • **Angiotensin II** raises Blood pressure in 2 ways: **Angiotensin II** = **Potent vasoconstrictor** Stimulates secretion of **Aldosterone** → increases Reabsorption of **Sodium** and **Water** by kidneys • Water absorption increases total blood volume → increases blood pressure	
Renal cortex and outer medulla	**Erythropoietin**
• Stimulates production of RBC in bone marrow (**Erythropoiesis**) • Stimulated when the amount of oxygen delivered to kidneys is lower than normal	

2. From Placenta

Location	Hormones secreted
Chorionic villi	**Estradiol, Progesterone, Human Chorionic Gonadotropin (HCG), Human Chorionic Somatomammotropin (HCS)**

- HCG = maintains corpus luteum for continued secretion of Progesterone =maintains pregnancy
- HCS = stimulates development of mammary glands for lactation

3. From Heart

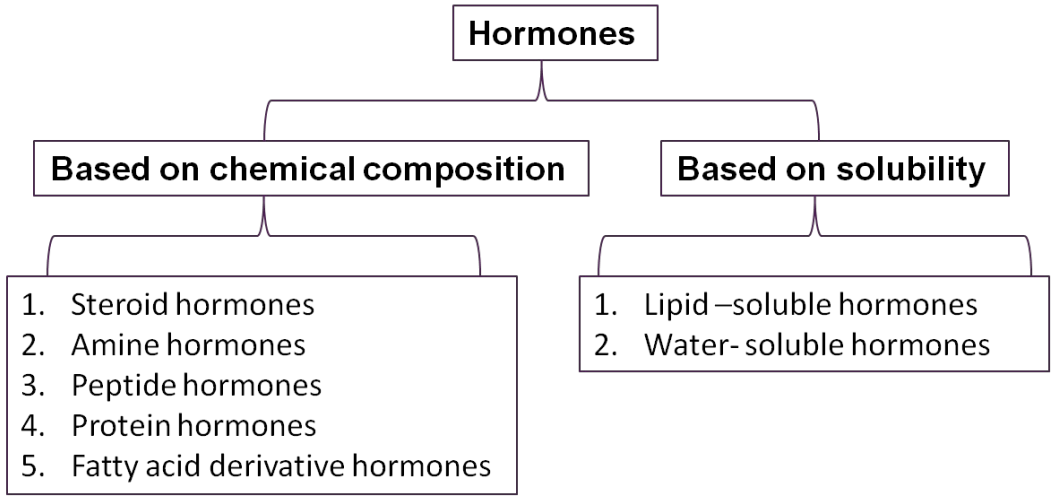
Location	Hormones secreted
Atrial wall	**Atrial Natriuretic factor (ANF)**

- Decreases blood pressure
- When BP (high) = ANF secreted = dilation of Blood vessels = reduces BP

4. From GI tract

Location	Hormones secreted
G- cells of Stomach and Duodenal mucosa	**Gastrin**
• Stimulates secretion of **Hydrochloric acid** and **Pepsinogen** • Also increases movements of stomach	
S-cells of duodenal and Jejunum mucosa	**Secretin**
• Increases secretion of **bicarbonate** by duct cells of pancreas and biliary tract • Decreases **gastric acid secretion** • Augments the action of **CCK** in producing pancreatic secretion of digestive enzymes	
I-cells of mucosa of upper small intestine	**Cholecystokinin**
• Stimulation of pancreatic enzymes secretion • Contraction of gallbladder • Relaxation of **sphincter of oddi** (which allows both bile and pancreatic juice to flow into the intestinal lumen)	
K-cells of Mucosa of duodenum	**Gastric inhibitory peptide**

eneral Mechanisms of Hormone action



Water - soluble hormones

- These hormones dissolve easily in water and blood plasma
- Cannot pass directly through the cell membrane
- Binds to receptors on the cell surface
- Usually produce rapidly but short-lived effects
- Ex. **Insulin, Glucagon, Growth Hormones (GH)**

Lipid - soluble hormones

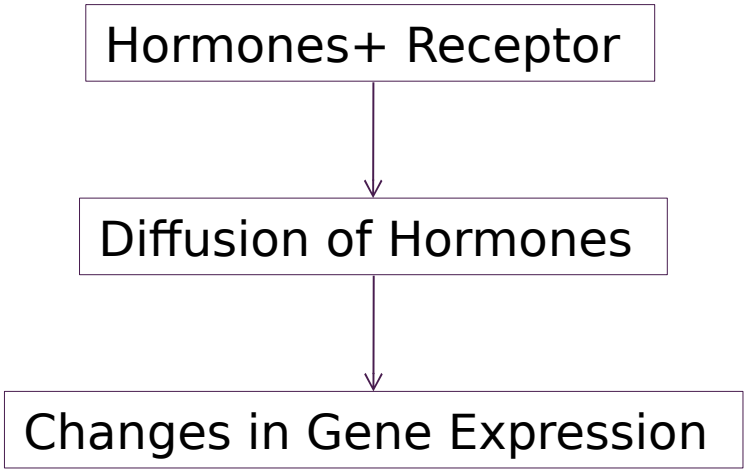
- Dissolves in fats and lipids but not easily in water
- Travel in blood attached to carrier proteins can pass through the cell membrane binds to receptors inside the cell (cytoplasm/ nucleus)
- Usually product slowly but long-lasting effects
- Ex. **Testosterone, Estrogen, Progesterone,**

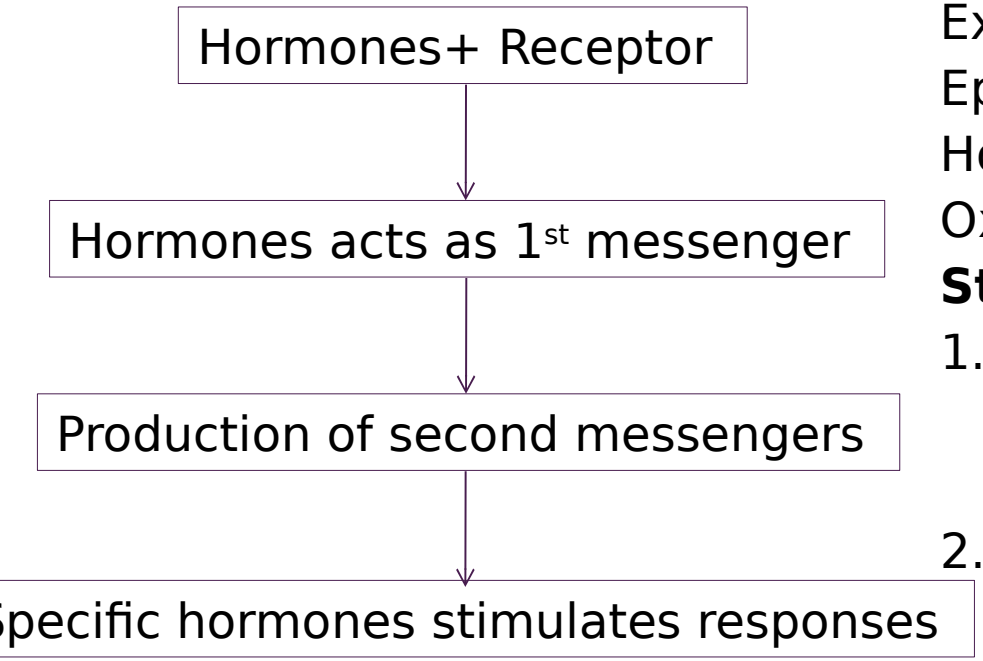
Action of lipid-soluble hormones

Examples of lipid soluble hormones are- Steroid hormones, Thyroid hormones

Steps of action:

1. Hormones bind to receptor within target cells
2. Hormones diffuse through lipid bilayer of the plasma membrane into cell
3. Hormones bind to and activates receptors located within the cytosol/ nucleus
4. Causes changes in Gene expression





Action of water- soluble hormones

Examples of water soluble are- Epinephrine, Insulin, Human Growth Hormones, Antidiuretic Hormone, Oxytocin and Glucagon

Steps of action:

1. Hormones bind to receptors that are present on target cells surface
2. It acts as a first messenger (hormone)
3. Production of second messenger (**cAMP, cGMP, diacylglycerol, inositol triphosphate, Calcium** etc.)
4. Specific hormone- stimulates responses happens

Hormones and Diseases

Hormonal disorders are caused by,

- Inadequate release (**Hyposecretion**)
- Excessive secretion (**Hypersecretion**)
- **Defective** hormone receptors
- **Inadequate** number of hormone receptors
- **Defective** second messenger systems

Rashmi MG

Pituitary gland disorder

Growth hormone related:

Hyposecretion-

Pituitary dwarfism

Symptoms- Short stature, slow growth during childhood, delayed puberty

Prevention- early diagnosis; growth hormone replacement therapy, balanced nutrition and regular medical monitoring

Hypersecretion-

Gigantism (child) and Acromegaly (adult)

Symptoms- excessive height and rapid growth, enlarged head and feet (Gigantism); enlarged hands, feet, jaw and nose, thickened skin, joint pain

Figure 76-9. Patient with acromegaly. *(Courtesy of Dr. Vishnu Garla.)*

Source: John E Hall, Michael E Hall (2021), Guyton and Hall Textbook of Medical Physiology, Elsevier publications, 14th Edition, Page no.888

FIGURE 56.12
The Alton giant. This photograph of Robert Wadlow of Alton, Illinois, taken on his 21st birthday, shows him at home with his father and mother and four siblings. Born normal size, he developed a growth hormone–secreting pituitary tumor as a young child and never stopped growing during his 22 years of life.

Source: Raven Johnson, Biology, 6th Edition, Page

Antidiuretic hormone related

Defective in ADH receptor – *Diabetes insipidus*

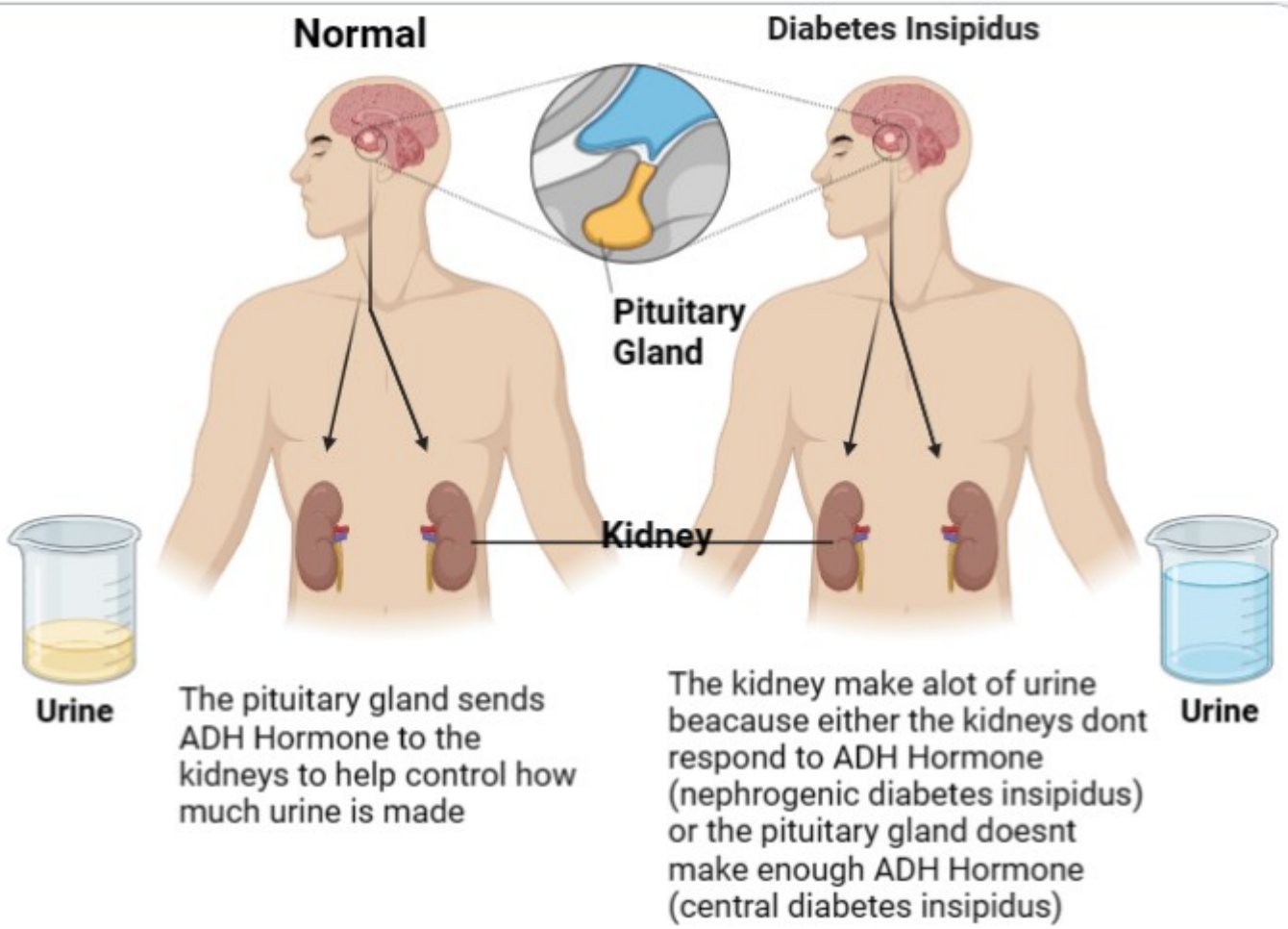
Symptoms- excessive urination, excessive thirst, dehydration, dry skin and fatigue

Prevention- adequate water intake, regular monitoring of fluid balance

Hypersecretion- ***Concentrated urine***

Symptoms- very concentrated urine, reduced urine output, water retention,

Diabetes Insipidus


Normal
Diabetes Insipidus
Pituitary Gland
Kidney
Urine
The pituitary gland sends ADH Hormone to the kidneys to help control how much urine is made
The kidney make alot of urine beacause either the kidneys dont respond to ADH Hormone (nephrogenic diabetes insipidus) or the pituitary gland doesnt make enough ADH Hormone (central diabetes insipidus)
Urine

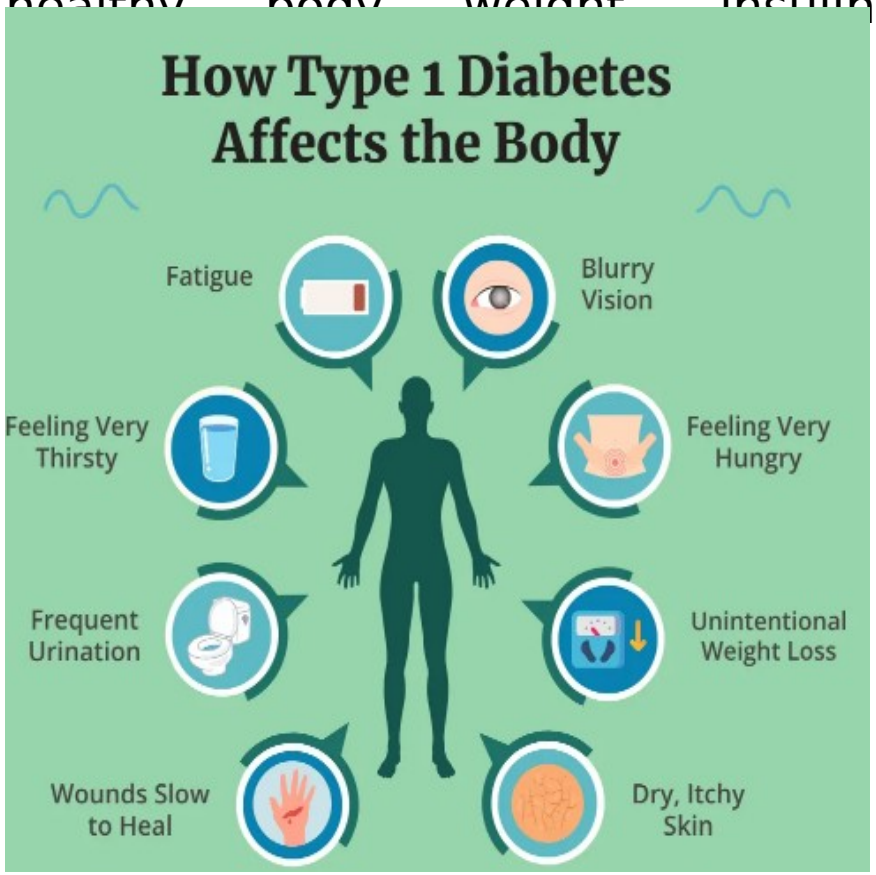
Source: https://www.biorender.com/template/diabetes-insipidus

Pancreatic islet disorders

Hyposecretion of Insulin- *Type 1 Diabetes*

Symptoms- high blood glucose level, frequent urination, increased hunger, weight loss and fatigue

Prevention- healthy diet, maintain healthy body weight, insulin

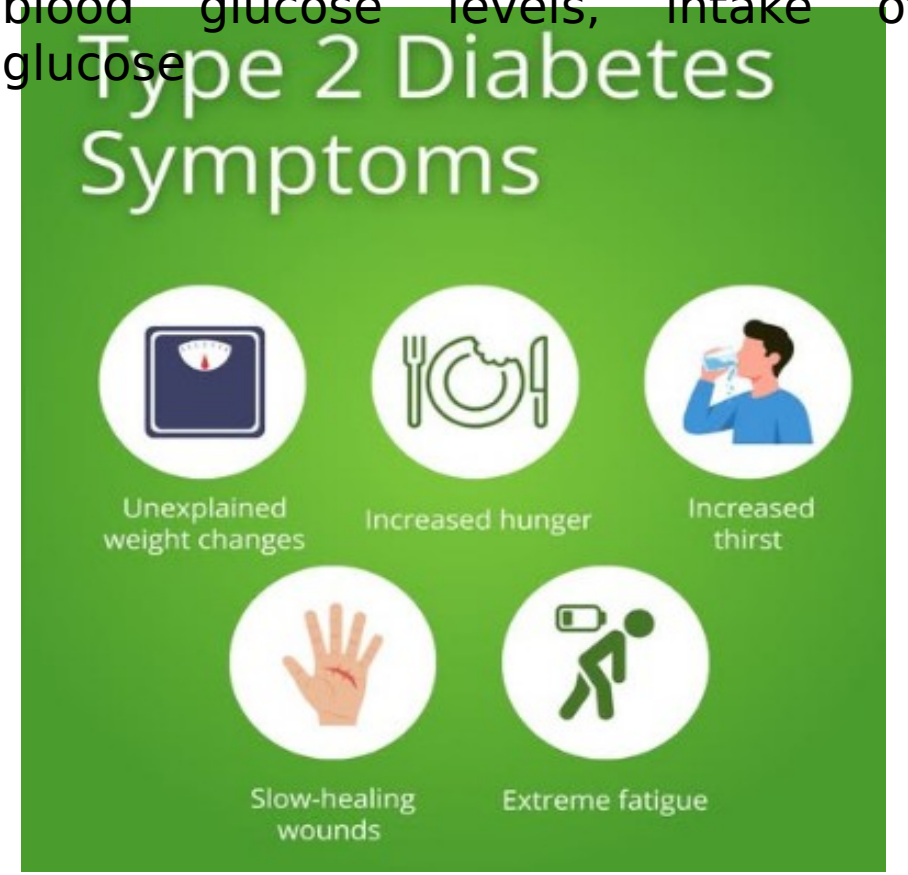


Source: https://www.everydayhealth.com/type-1-diabetes/guide/physical-changes/

High insulin resistance-*Type 2 diabetes*

Symptoms- low blood sugar, sweating, dizziness, trembling, confusion and weakness

Prevention- regular meals, monitor blood glucose levels, intake of glucose



Source: https://www.facebook.com/OzarksHealthcare/posts/type-2-diabetes-can-be-subtle-but-the-impact-is-serious-knowing-the-signs-and-ac/1506859384773897/

Rashmi MG

Thyroid gland disorders

Hyposecretion of thyroxin- ***Hypothyroidism***

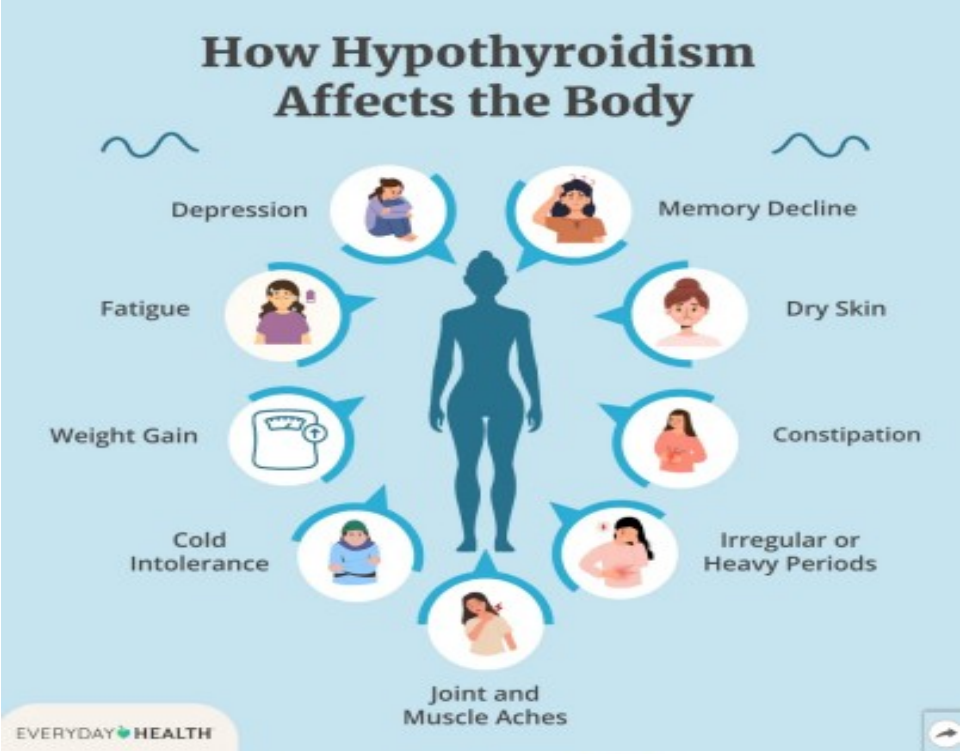
Symptoms- Fatigue, weight gain, slow heart rate, dry skin, hair loss

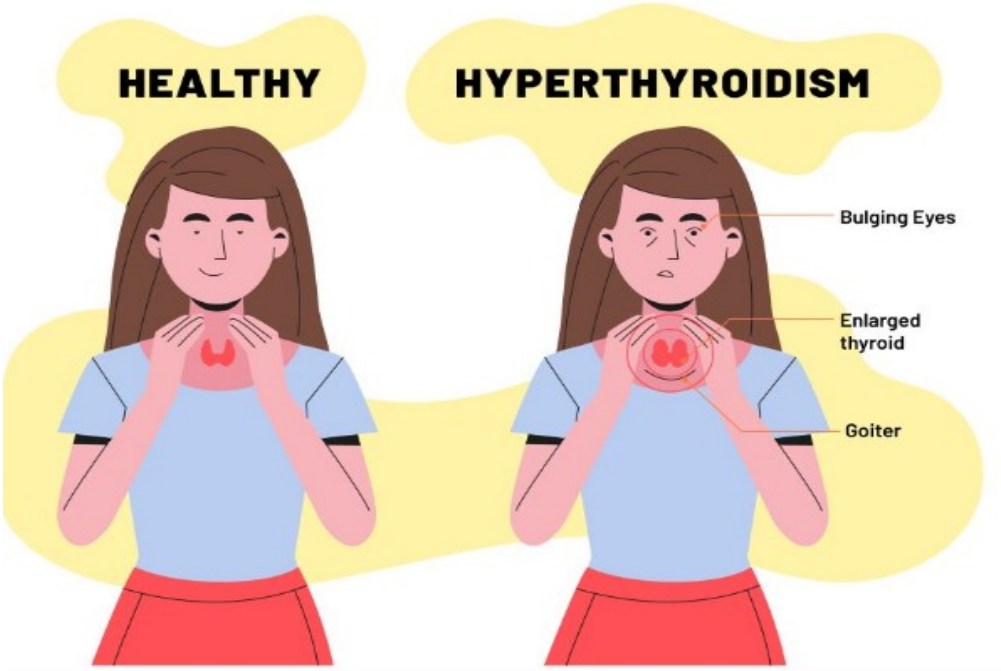
Prevention- adequate iodine intake, regular thyroid check-ups, thyroxine replacement therapy

Hypersecretion of thyroxine- ***Hyperthyroidism***

Symptoms- weight loss, rapid heartbeat, increased appetite, nervousness and irritability, bulging eyes

Prevention- anti-thyroid medications, surgery/ radioactive therapy





Source:
https://www.everydayhealth.com/thyroid-conditions/hypothyroidism/overview/
https://www.omegahospitals.com/blog/hyperthyroidism/

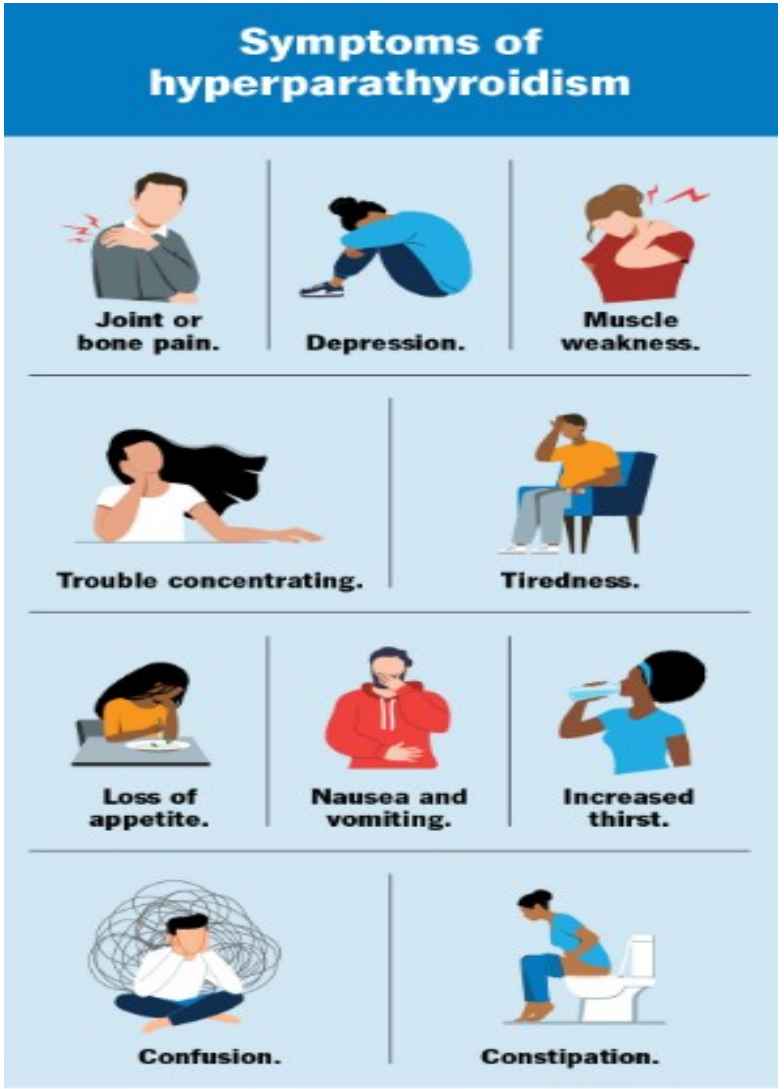
Parathyroid gland disorders

Low concentration of blood calcium- *Hypoparathyroidism*

Symptoms- muscle cramps and spasms, tingling in fingers and toes, seizures, weakness and fatigue

Prevention- adequate calcium and vitamin D intake, regular

Some symptoms of hypoparathyroidism
Muscle cramps and spasms
"Brain fog" or confusion
Abnormal heart rhythm
Tingling or numbness in your lips, fingers and toes
Brittle nails and dry hair or skin
Cleveland Clinic

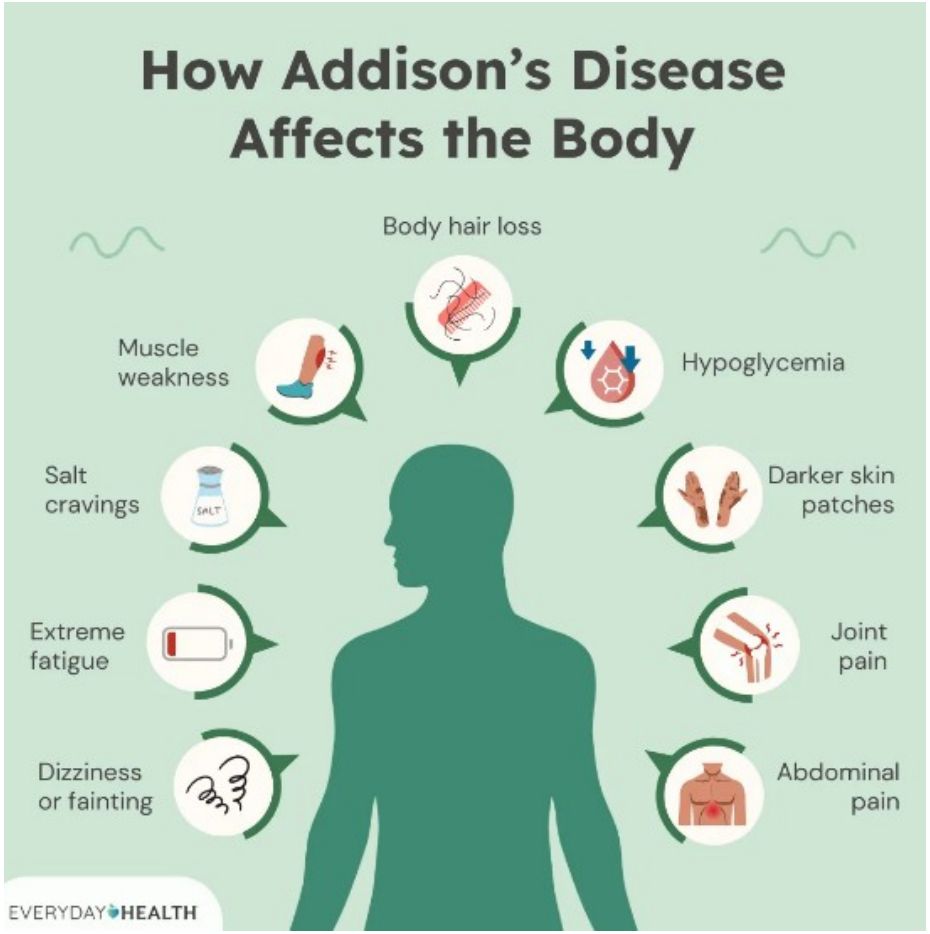


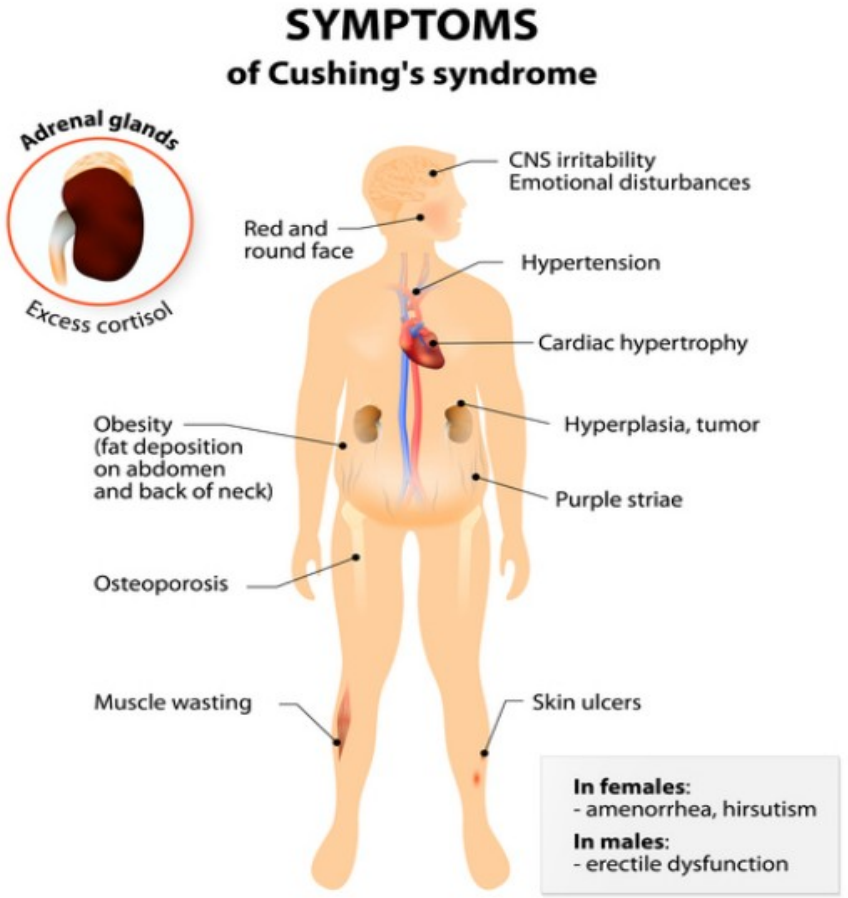
High concentration of blood calcium- *Hyperparathyroidism*

Symptoms- weak bones, fractures, kidney stones, excessive thirst and urination, muscle weakness

Prevention- adequate hydration, surgical

ource: https://my.clevelandclinic.org/health/diseases/22672-hypoparathyroidism
https://my.clevelandclinic.org/health/diseases/14454-hyperparathyroidism





Adrenal gland disorders

Hyposecretion of Glucocorticoids- *Addison's disease*

Symptom- extreme fatigue, weight loss, low blood pressure, muscle weakness, darkening of skin

Prevention- hormone replacement therapy, proper management of stress and

Source: https://www.everydayhealth.com/autoimmune-diseases/addisons-disease/
https://medlineplus.gov/genetics/condition/cushing-disease/

Hypersecretion of Cortisol - *Cushing's syndrome*

Symptoms- weight gain (especially around abdomen and face), high blood pressure, muscle weakness, thin skin with easy bruising

Prevention- treat adrenal or pituitary tumors, avoid unnecessary long-term steroid use, regular monitoring and